STUDY ON C REACTIVE PROTEIN-AN AID FOR ASSESSING AND MONITORING THE SEVERITY OF ACUTE PANCREATITIS IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM

Dissertation submitted to

THE TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY

CHENNAI – 600 032

In partial fulfillment of the regulations
For the awards of the degree of
M.S. DEGREE BRANCH – I
GENERAL SURGERY

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE, SALEM

APRIL 2017

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation entitled **"STUDY ON C REACTIVE PROTEIN-AN AID FOR ASSESSING AND MONITORING THE SEVERITY OF ACUTE PANCREATITIS IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM"** is a bonafide work done by DR.S.T.JAYASUDHA under my guidance during the period of 2015-2016. This has been submitted to the partial fulfillment of the award of M.S. degree in General Surgery (Branch I) Tamil Nadu Dr. M.G.R. Medical University, Chennai-32.

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DECLARATION

I solemnly declare that this dissertation **"STUDY ON C REACTIVE PROTEIN-AN AID FOR ASSESSING AND MONITORING THE SEVERITY OF ACUTE PANCREATITIS IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM.** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem-636030 under the guidance and supervision of PROF. DR.K.KESAVALINGAM., PROFESSOR OF GENERAL SURGERY, Govt. Mohan Kumaramangalam Medical College and Hospital, Salem. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in fulfillment of the University regulations for the award of the degree of M.S. General Surgery (Branch I).

Place: Salem

Date: 3.10.2016

DR.S.T.JAYASUDHA

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Ethical Committee Meeting held on 18.06.2015 at 10.00 A.M in the Seminar Hall, IInd Floor, Medicine Block, Govt. Mohan Kumaramangalam Medical College Hospital, Salem 01.

The following Members were attended the Meeting.

MEMBERS:

1. Dr. V. Dhandapani, MD., Deputy Chairman, External Social Scientist, ECIRB.
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Sl. No.	Name of the Presenter with Address	Title	Name of the Guide and Address	Whether it is Approved or not.
1.	Dr. S.T. Jayasudha, II Year, Post Graduate Student of MS (General Surgery), GMKMC, Salem-30.	"Study on C Reactive protein – An aid for assessing and monitoring the severity of acute pancreatitis in GMKMCH, Salem From June 2015 to June 2016"	Dr. K. Shanthi, MS., Associate Professor of General Surgery, GMKMC, Salem.	Approved

The Ethical Committee examined the studies in detail and is pleased to accord Ethical Committee approval for the above Post Graduate student of this College to carry out the studies with the following conditions.

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7. She should not claim any funds from the institution while doing the work or on completion.
8. She should understand that the members of IEC have the right to monitor the work with prior intimation.

To,
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25/06/15

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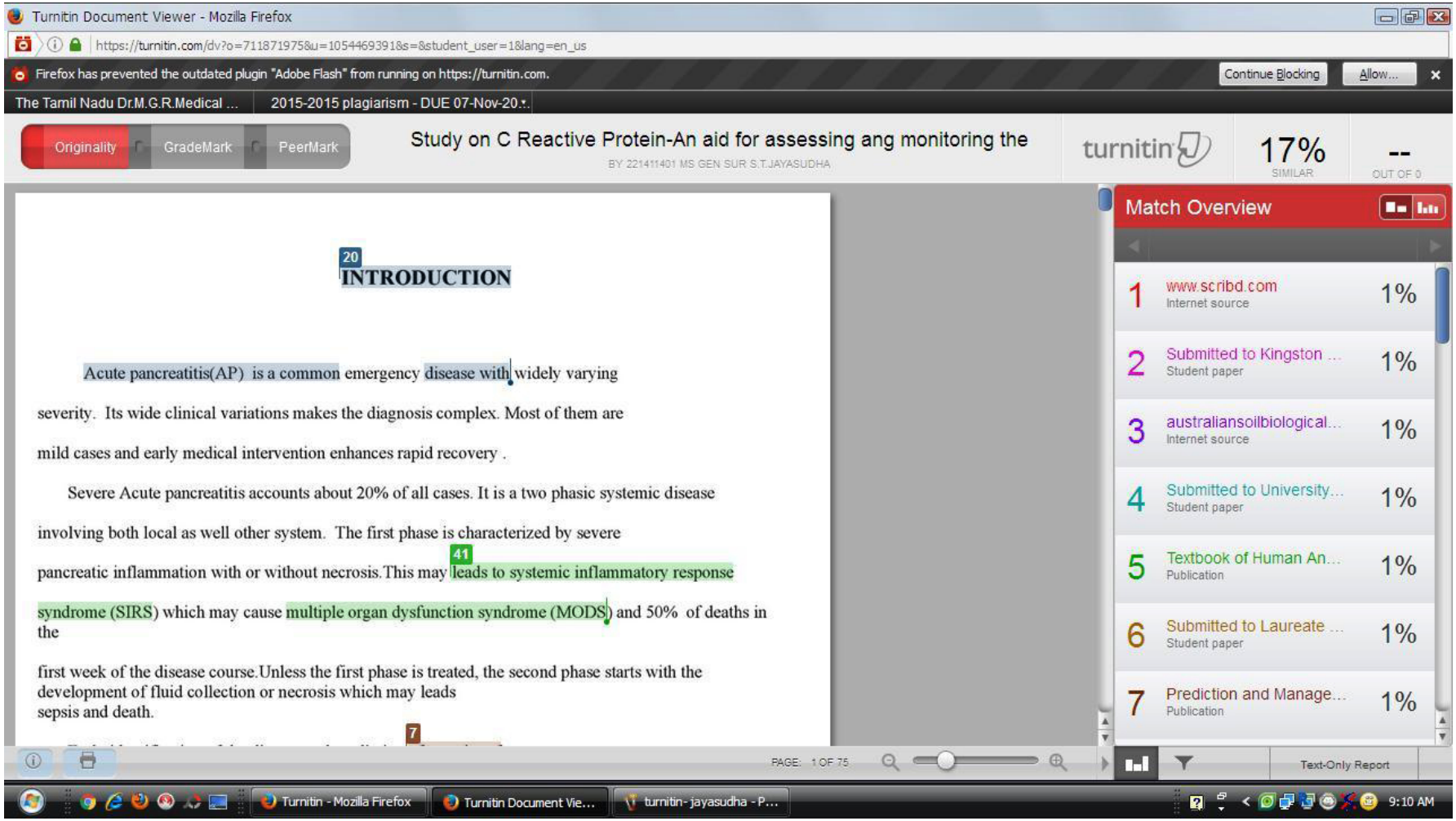
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INTRODUCTION
Acute pancreatitis(AP) is a common emergency disease with widely varying severity. Its wide clinical variations makes the diagnosis complex. Most of them are mild cases and early medical intervention enhances rapid recovery .
Severe Acute pancreatitis accounts about 20% of all cases. It is a two phasic systemic disease involving both local as well other system. The first phase is characterized by severe pancreatic inflammation with or without necrosis.This may 41 leads to systemic inflammatory response syndrome (SIRS) which may cause multiple organ dysfunction syndrome (MODS) and 50% of deaths in the first week of the disease course.Unless the first phase is treated, the second phase starts with the development of fluid collection or necrosis which may leads sepsis and death.
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ABSTRACT

BACKGROUND

Acute pancreatitis (AP) is the most terrible of all the calamities that occur in connection with the abdominal viscera. The suddenness of its onset, the illimitable agony which accompanies it, and the mortality attendant upon it, all render it the most formidable of catastrophies.

Acute pancreatitis is a common emergency disease with widely varying severity. Its wide clinical variations make the diagnosis complex. Mostly, only mild cases and conservative treatment results in rapid recovery. Severe disease constitutes 15–20% of all cases with frequent involvement of regional tissues and remote organ systems as two phase systemic disease. In the first phase extensive pancreatic inflammation and/or necrosis are followed by a systemic inflammatory response syndrome (SIRS) that may lead to multiple organ dysfunction syndrome (MODS) within the first week. About 50% of deaths occur during the first week of the attack, mostly from MODS. Unless the first phase is treated, the second phase ensues after the second week of onset and includes the development of infected pancreatic necrosis or fluid collection with possible progression to overt sepsis, MODS and death. Early identification of acute pancreatitis and especially detection of severe form of the disease is very important.

Currently, the diagnosis of acute pancreatitis is based on measurements of serum amylase and/or lipase activity, which are considered unsatisfactory due to their low level of accuracy. There are a number of well known measurements for evaluating the prognosis of acute pancreatitis, such as the RANSON, GLASCOW, BISAP and Acute Physiology and Chronic Health Evaluation (APACHE II). All of these require measurement of many clinically-based parameters and are very complicated and time consuming. In this study, usefulness of C-reactive protein to assess and monitor the severity of acute pancreatitis was evaluated.

OBJECTIVES

The primary objective is study on C-reactive protein for assessing and monitoring the severity of acute pancreatitis.

METHOD

This is a prospective study, where 75 patients admitted to our hospital with acute pancreatitis, who met with the inclusion and exclusion criteria, were subjected to clinical examination and relevant investigations such as serum amylase, serum lipase, USG abdomen, serial monitoring of C-reactive protein. The results are evaluated and analyzed by comparing with serial monitoring of alpha 1 antitrypsin. White cell

count, erythrocyte sedimentation rate, temperature were used as reference data.

RESULTS

In our study of 75 patients of acute pancreatitis, 39 patients were found to have mild disease and 36 patients were found to have severe acute pancreatitis according to Atlanta criteria 2012. Etiologies of the disease were alcoholic, biliary and idiopathic.

Samples for CRP, Alpha 1 Antitrypsin, WBC, and ESR were collected on day1 of admission and on days 3, 5, 7, 9, 11 after admission. Temperature, ESR, Alpha1Antitrypsin values didn't discriminate acute pancreatitis as mild and severe disease. Although those values were high in severe acute pancreatitis, mean 95% confidence limits of mild and severe attacks were overlapped throughout the study.

On day 1 of admission, difference in WBC count between mild and severe disease, helps to discriminate between the two. As the disease progressed, CRP values reaches maximum in the end of first week, in severe acute pancreatitis and it takes more time to fall towards normal value. Hence CRP helped to differentiated between mild and severe acute pancreatitis better than WBC and Alpha 1 antitrypsin value. High level of CRP (>100mg /l) at first week suggests that patients who have

the disease requires 2 or more weeks to recover and there is risk of developing pancreatic collection.

Increased values of CRP reflect severe local inflammation in mild disease with benign clinical course.

Hence, CRP is a sensitive indicator of continuing inflammation and it may be of better value in selecting the cases who are more prone for developing high risk complications.

CONCLUSION

Temperature, ESR, Alpha1Antitrypsin values didn't discriminate acute pancreatitis as mild or severe disease. Although those values found high in severe acute pancreatitis, mean 95% confidence limits of mild and severe attacks were overlapped throughout the study.

Of the inflammatory markers studied, CRP was able to differentiate acute pancreatitis into mild and severe forms with greatest precision.

KEY WORDS

C - reactive protein, Alpha 1 Antitrypsin, Erythrocyte sedimentation rate, Acute pancreatitis, Severity.

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ABBREVATIONS

AP	-	Acute pancreatitis
CRP	-	C Reactive Protein
SIRS	-	systemic inflammatory response syndrome
MODS	-	multiple organ dysfunction syndrome
PSTI	-	pancreatic secretory trypsin inhibitor
SPINK1	-	serine protease inhibitor Kazal type 1
IL	-	Interleukin
MOF	-	Multiorgan failure
ARDS	-	Acute respiratory distress syndrome
AA	-	Alpha 1 anti trypsin
APACHE	-	Acute Physiology And Chronic Health Evaluation
SOFA	-	Sequential Organ Failure Assessment
BISAP	-	Bedside Index for Severity of Acute Pancreatitis
MAP	-	Mean Arterial Pressure
GCS	-	Glasgow Coma Scale
CT	-	Computerised Tomography
CECT	-	Contrast Enhanced Computerised Tomography
MRCP	-	Magnetic Resonance Cholangiopancreatography
ERCP	-	Endoscopic retrograde cholangiopancreatography
ICU	-	Intensive Care Unit

THE KEY TO MASTER CHART

M	–	Male
F	–	Female
IP No.	–	Inpatient Number
DOA	–	Date of Admission
ESR	–	Erythrocyte Sedimentation Rate
1	–	Mild Acute Pancreatitis
2	–	Severe Acute Pancreatitis
Temperature	–	Measured in Farenheit
ESR	–	Measured in millimeters per hour
AA	–	Alpha 1 Antitrypsin measured in grams per litre
CRP	–	C- Reactive Protein measured in milligrams per litre
WBC	–	White Blood Count cells per cubic millimeter of blood
Mg/Ltr	–	Milligram per Litre
g/Ltr	–	Gram per Litre

INTRODUCTION

Acute pancreatitis (AP) is a common emergency disease with widely varying severity. Its wide clinical variations make the diagnosis complex. Most of them are mild cases and early medical intervention enhances rapid recovery. Severe Acute pancreatitis accounts about 20% of all cases. It is a two phasic systemic disease involving both local as well other system. The first phase is characterized by severe pancreatic inflammation with or without necrosis. This may leads to systemic inflammatory response syndrome (SIRS) which may cause multiple organ dysfunction syndrome (MODS) and 50% of deaths in the first week of the disease course. Unless the first phase is treated, the second phase starts with the development of fluid collection or necrosis which may leads sepsis and death.

Early identification of the disease and prediction of severity of the acute pancreatitis is very important. There are different scoring systems available for the early assessment of AP. Most widely used scoring systems are RANSON, BISAP and APACHE. They are cumbersome and difficult to use in clinical practices because of their multifactorial nature.

Unifactorial prognostic indices such as C - reactive protein (CRP), serum amylase, serum lipase and alpha-1 antitrypsin are easy to obtain in

normal practice. Several research people evaluated CRP as predictor for severity of acute pancreatitis and found CRP differentiates mild and severe forms of the acute pancreatitis with greatest precision and is highly sensitive in detecting necrotic forms of AP.

In this study, we evaluated C Reactive Protein (CRP) as a marker for assessing and monitoring the severity of acute pancreatitis and the collected data were compared with the values given by the white cell count, erythrocyte sedimentation rate, temperature and by alpha 1 antitrypsin. We analyzed the data and found that CRP value was high (100 mg/dl) at the end of the first week of acute pancreatitis and serial high CRP levels in serum is a definite indicator of severity of the acute pancreatitis than white cell count, erythrocyte sedimentation rate, temperature and alpha 1 antitrypsin.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

- Herophilus (335–280 BC) was the first person identified the pancreas. A few hundred years after, Rufus named the pancreas.
- In 1642-Johann George Wirsung, a Physician in Germany, discovered Main pancreatic duct (Wirsung's duct).
- In 1724, Giovanni Domenico Santorini dissected accessory pancreatic duct (Santorini's duct).
- In 1833, Anselme payen isolated amylase, a enzyme present in exocrine pancreas.
- In 1893, Gustav-Edouard Laguesse given the name Islets of Langerhans as an honour of Paul Langerhans.
- In1922, Frederick Banting and John Macleod discovered insulin and win the Nobel prize in 1923.Banting shared his part of the prize money with a younger coworker Charles Best.
- In 1953, Staub Sinn and Behrens purified Glucagon, otherwise known as "hyperglycemic glycogenolytic factor".
- 1965, Axelssor and Laurell investigated the allelic variants of Alpha 1 Antitrypsin causing disease.

EMBRYOLOGY OF PANCREAS

Septum transversum produces two pancreatic buds at the junction of foregut and midgut at fifth weeks of gestation from endodermal lining of duodenum distal to future stomach. The larger dorsal bud grows rapidly than the smaller ventral bud. The ventral bud rotates towards the dorsal bud as it is carried by common bile duct. Inferior part of head of the pancreas and uncinate process develops from the ventral bud. Dorsal bud gives rise to head, whole of neck, body and tail of this organ. The major pancreatic duct forms from ventral bud and terminal part of dorsal bud, whereas the accessory pancreatic duct forms from proximal part of dorsal bud.

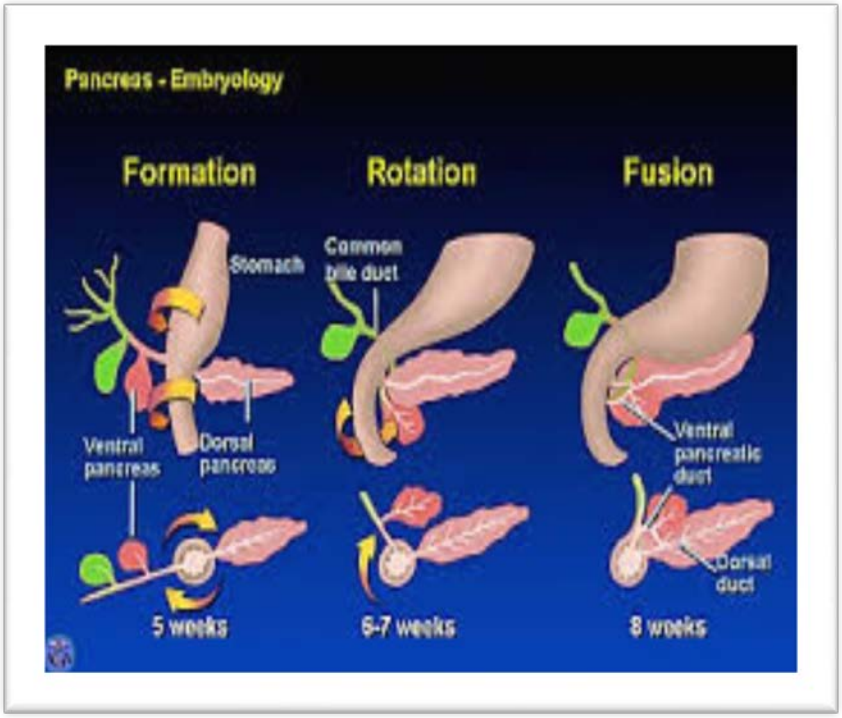


Fig. 1. Embryology of Pancreas.

DEVELOPMENTAL ANOMALIES OF THE PANCREAS

1. **Pancreas divisum** is one of the congenital defect in which there is no fusion of ventral and dorsal pancreatic ducts. There are three variants of pancreatic divisum.
    a. Type 1 (Classical divisum) where there is total failure of fusion.
    b. Type 2 where dorsal drainage is dominant in the absence of the duct of Wirsung.
    c. Type 3 (Incomplete divisum) where a small communicating branch is present

    This is associated with recurrent acute pancreatitis because of inadequate drainage of pancreatic secretions via the minor papilla.
2. As a results of failure of the ventral bud to rotate with the duodenum producing an **annular pancreas**, encircling the second part of the duodenum.

    Annular pancreas may be the cause of duodenal obstruction in neonatal period. In adults, it is asymptomatic and discovered incidentally. It may be the cause of gastric outlet obstruction, acute or chronic pancreatitis or peptic ulcer bleeding. Ectopic pancreas is found mostly at gastric antral sub mucosa, proximal

part of the duodenum or the jejunum. This is usually asymptomatic or symptomatic when complications such as ulceration, bleeding develops.

3. Absence of the ventral or dorsal pancreatic buds results in **Pancreatic agenesis.**
4. **Accessory pancreatic tissue** will be present in duodenal wall, jejunum ,ileum or Meckel's diverticulum.
5. **Inversion of pancreatic ducts**: In this condition, the accessory duct is larger than the main duct, and the main drainage of the pancreas is through the minor duodenal papilla

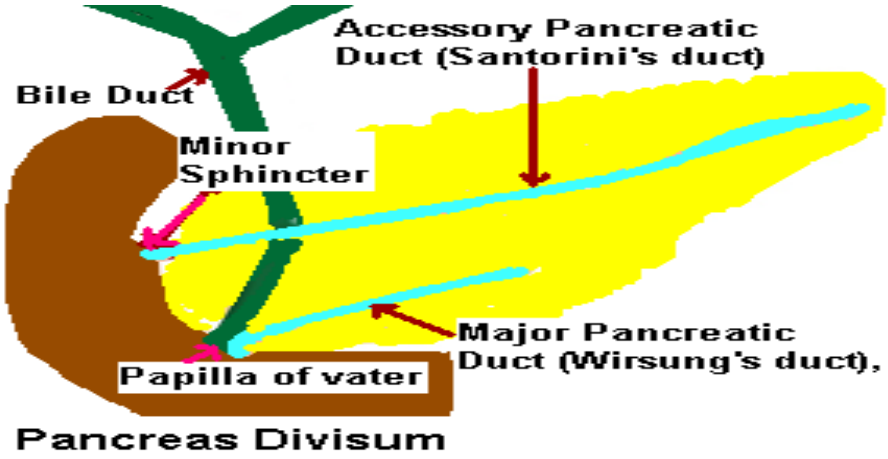


Fig. 2. Pancreas divisum

ANATOMY

The pancreas (pan = all; kreas = flesh in Latin), originally means sweetbread, is both exocrine and endocrine soft, lobulated retroperitoneal organ located in left upper part of the abdomen obliquely behind stomach at the level of first and second lumbar vertebrae extending from the C-loop of the duodenum to the splenic hilum (Fig 3).The pancreas is about 15-20 cm long, 2.5-3.8 cm broad and 1.2-1.8 cm thick and weighs about 75 to 100g.

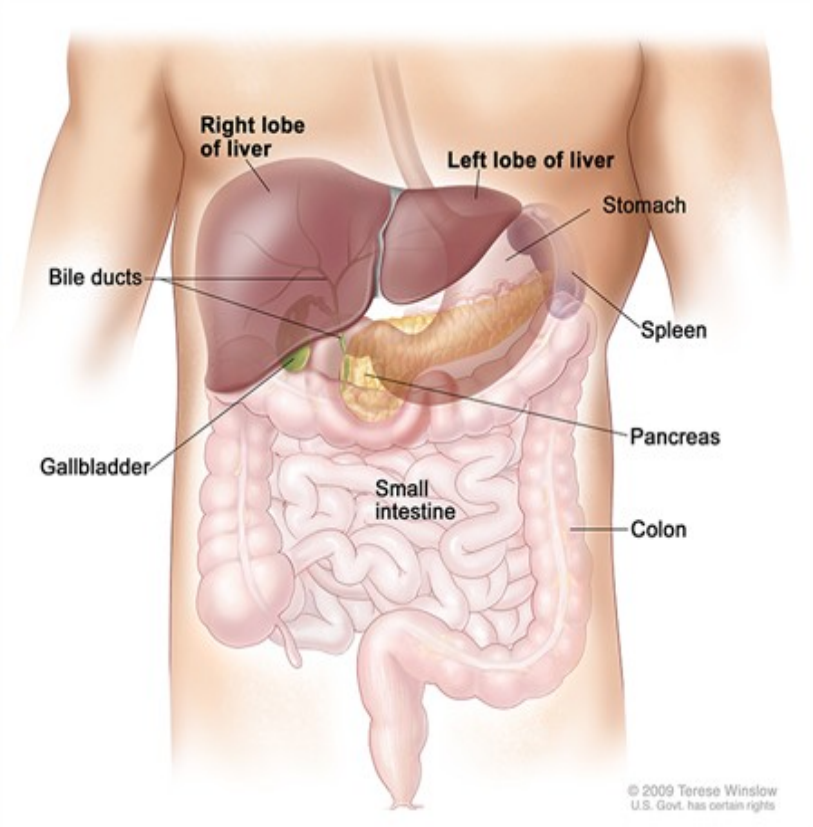


Fig. 3.Location of Pancreas.

Anatomically, the pancreas is divided into four parts

1.**Head**

2.**Neck**

3.**Body and**

4.Tail

1.Head of the pancreas

Head is situated within the duodenal curvature. Head has two surfaces (anterior and posterior), three borders, (superior, inferior and right lateral),one process, named the **uncinate process**, which projects to the left arising from lower part of the head.

The anterior surface relations are gastro duodenal artery, transverse colon and jejunum from above downwards. The relations of posterior surface are inferior vena cava, renal veins, right crus of the diaphragm, and bile duct. Superior border overlaps first part of pancreaticoduodenal artery. The inferior border is related to inferior pancreaticoduodenal artery and to third part of duodenum. Right lateral border related to terminal bile duct, duodenum at the second part, and anastomosis between the two pancreaticoduodenal arteries.

Uncinate process is related to the superior mesenteric vessels anteriorly, and to the aorta posteriorly.

2.Neck

The neck measures of length 2.5 cm and lies between head and body. It is directed upwards and forwards at first, and then upwards and to the left, with two surfaces, anterior and posterior. The relations of anterior surface are the pylorus and the peritoneum covering the posterior wall of the lesser sac. The gastroduodenal and superior pancreaticoduodenal arteries lie at its junction with the head. The relations of posterior surface are termination of the superior mesenteric vein and the beginning of the portal vein.

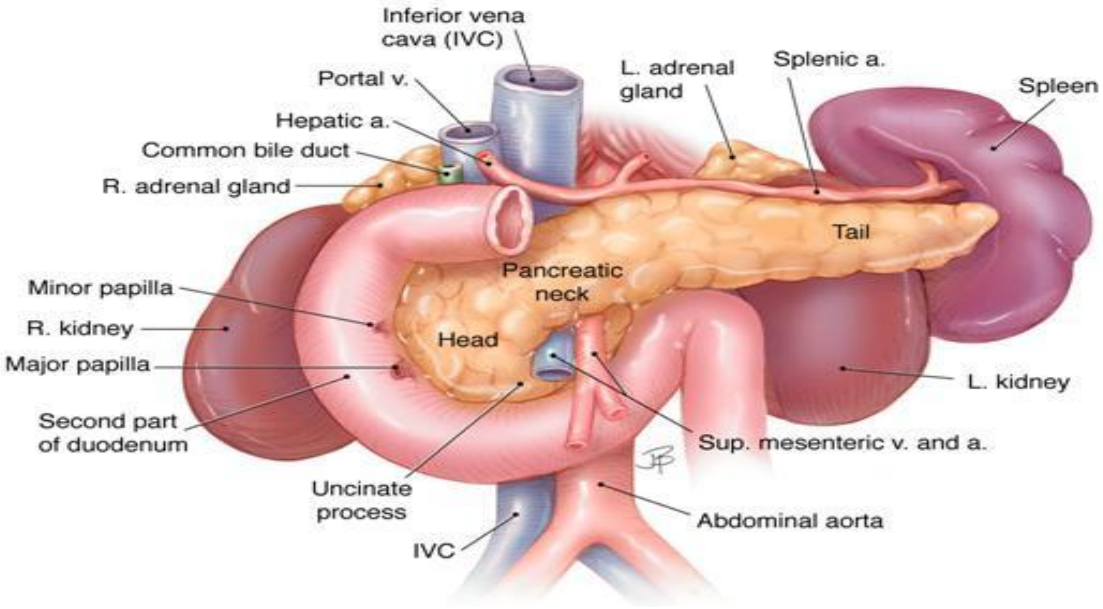


Fig 4.Parts and relations of Pancreas.

3.Body

The largest part of the pancreas is the body which is elongated and located at the level of transpyloric plane which inclines upward and backward slightly towards the left. A projection called the **tuber omentale** is a part of the body projects upwards beyond the lesser curvature of the stomach, and is related to the lesser omentum across the lesser sac.

It has three surfaces, anterior, posterior and inferior and three borders anterior, superior and inferior. The anterior surface is related to the stomach and to the lesser sac with peritoneal covering.

The posterior surface is devoid of peritoneum, and is related to the aorta with the origin of the superior mesenteric artery, left crus of the diaphragm, left suprarenal gland, left kidney, left renal vessels and splenic vein. The inferior surface is related to the duodenojejunal flexure, the left colic flexure, and the coils of jejunum and is covered by peritoneum. The root of the transverse mesocolon attached to the anterior border. The superior border is related to the hepatic artery to the right and the splenic artery to the left, coeliac trunk over the tuber omentale. The inferior border is related to the superior mesenteric vessels at its right side.

4. Tail of the Pancreas lies in the lienorenal ligament and ends by contacting with the gastric surface of the spleen.

Ducts of Pancreas

The secretions of exocrine pancreas drains into two ducts.

1. The **main pancreatic duct of Wirsung** with diameter of about 2 to 3mm begins at the tail receives numerous small tributaries at right angles to its long axis formig‘herring bone pattern’. It runs through the body and the neck, opens at the ampulla of Vater after joining with the common bile duct, which opens on the summit of the major duodenal papilla, on the medial aspect of the duodenum at the second part 8 to 10 cms distal to the pylorus.

2. The **accessory duct (Santorini duct**) begin in the lower part of the head, and open into the duodenum at the minor duodenal papilla which is situated 6 to 8 cm distal to the pylorus ventral to that of the main duct.

The sphincter of Oddi is the muscle fibers around the ampulla.

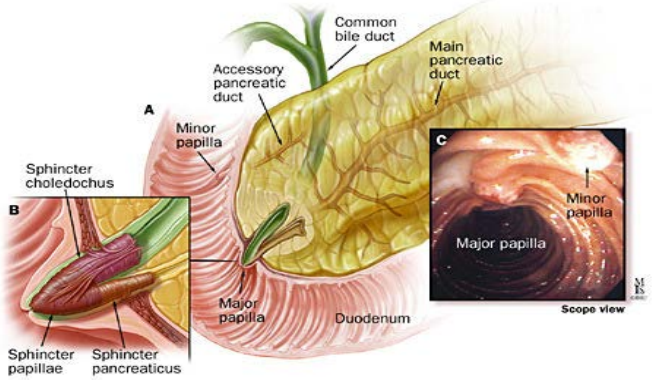


Fig. 5. A. Anatomy of the papilla; B. Sphincter; C.Endoscopic view.

BLOOD SUPPLY

The following are the arteries supplying the pancreas :

(1)Pancreatic branches of the splenic artery,

(2) Superior pancreaticoduodenal artery, and

(3)Inferior pancreaticoduodenal artery (Fig.6)

As the duodenum and the pancreas develops at the junction of the foregut and midgut, branches of both the coeliac and superior mesenteric arteries supplies it.

Venous drainage is by splenic vein, superior mesenteric vein and portal veins.

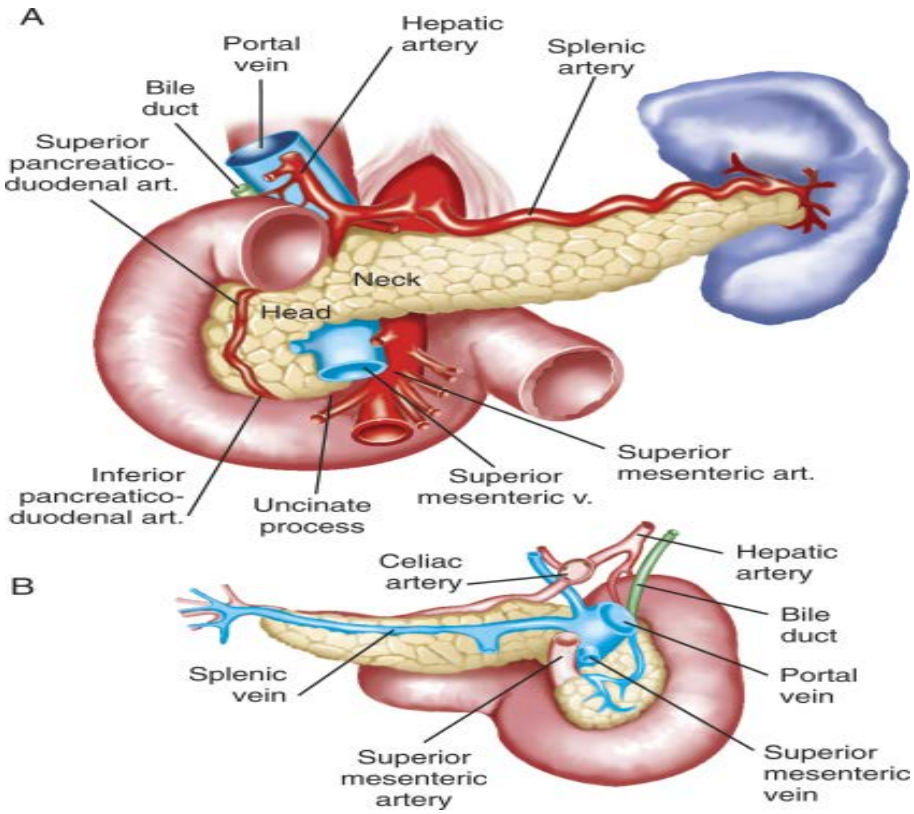


Fig 6.Blood supply of Pancreas.

LYMPHATIC DRAINAGE

Lymphatics follow the arteries and empties into the following groups of lymph nodes

1. Pancreaticosplenic,

2. Coeliac and

3. Superior mesenteric lymph nodes

NERVE SUPPLY

The vagus nerve which is parasympathetic and sympathetic splanchnic nerves supply the pancreas through the plexuses around its arteries. Parasympathetic nerve regulates pancreatic secretion. Sympathetic nerves are vasomotor.

HISTOLOGY

The pancreas is divided into lobules by connective tissue septae which is the invagination of capsule.

The exocrine portion of pancreas consists of acini and ducts. Acinus (Latin word meaning berry in a cluster) are the pear shaped cells lined by pyramidal cells with basal round nuclei, containing zymogen granules. These cells synthesis and secrete the digestive enzymes rich in alkaline bicarbonate ions. Pancreatic exocrine secretion from acini flow through ducts into the duodenum and are classified into four types.

1. **Intercalated ducts** have flattened cuboidal epithelium that extends up into the lumen of acinus to form centroacinar cells
2. **Intralobular ducts** have classical cuboidal epithelium and are seen within lobules. Secretions from intercalated ducts drain into it.
3. **Interlobular ducts** are found between lobules. The smaller ducts have cuboidal epithelium and the larger ducts have columnar epithelium. They receive secretion from intraocular ducts.
4. **Main pancreatic duct** receive secretion from interlobular ducts and penetrates through the wall of the duodenum.

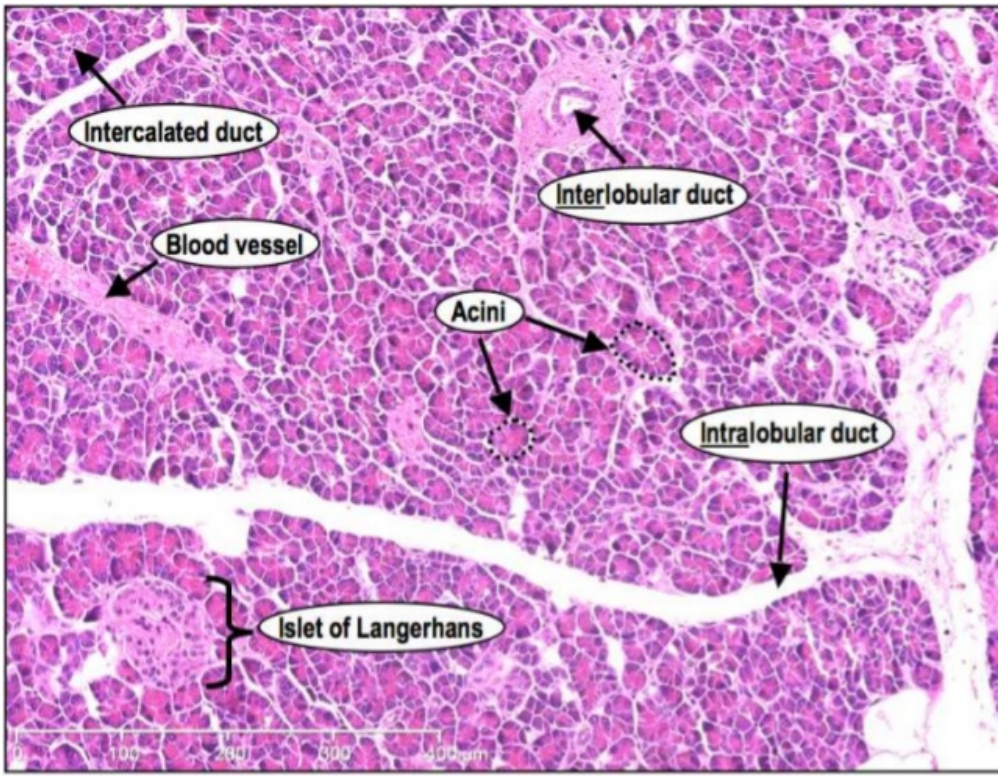


Fig.7.Histology of Pancreas.

Endocrine part consist of specialized clumps of secretory cells called Islets of Langerhans among acini, supported by reticulin fibres and delicate capsule and contains three major cell types.

Alpha cells with subtype A1 and A2. These are granular and acidophilic and form about 20% of the cell population. Al cells belong to enterochromaffin group and secrete pancreatic gastrin and serotonin. A2 cells secrete glucagon.

Beta cells which are granular and basophilic, forming about 80% of the cell population and produce insulin.

Delta cells secrete somatostatin. Islets are supplied by arterioles with branching fenestrated capillaries, into which hormones are secreted, drained by venules which pass into interlobular vein.

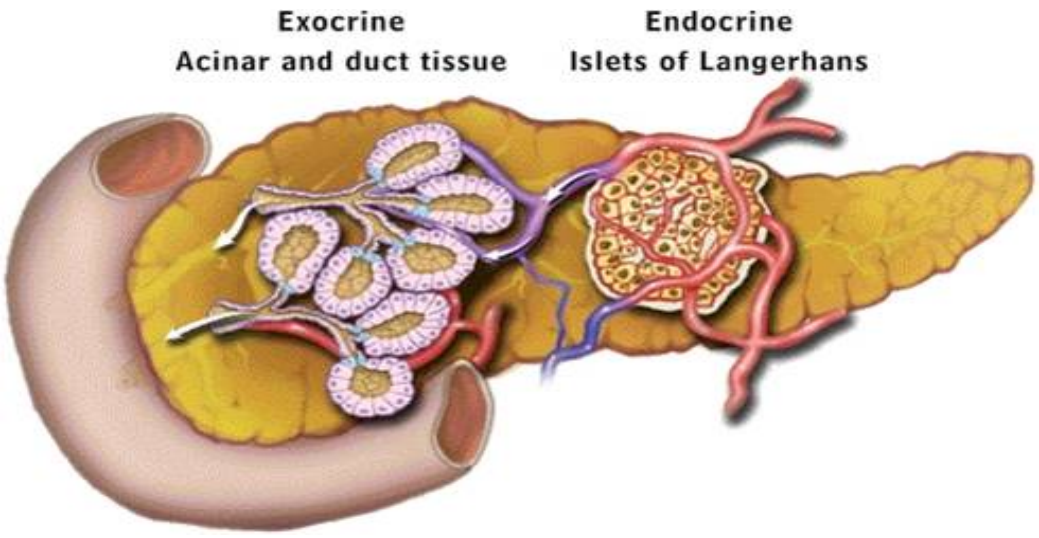


Fig.8.Exocrine and Endocrine Parts of Pancreas.

FUNCTIONS

1.Exocrine pancreas:

The pancreas produces 500 to 800 ml of colorless, odorless secretion per day. Secretion contains juice from acinar cell and duct cell mainly alkaline. Amylase, proteases, and lipases are the enzymes secreted by the acinar cell which helps in digestion of carbohydrates, protein and fat.

Amylase is the active form of the enzyme secreted in the pancreas, and it digest starch and glycogen to glucose. Lipase breaks down fat into fatty acids and glycerol.

The function of acinar cells is to synthesis and secrete the inactive digestive enzyme precursors (trypsinogen, proelastases, prophospholipase A2) into the duodenum.

Endoplasmic reticulum in the cell, synthesis zymogens and packed into secretory granules. Upon stimulation of acinar cells, granules discharge the contents into lumen of acinus by exocytosis and drains into duodenum via the ducts. In the duodenum, enterokinase catalyse the conversion of inactive trypsinogen to active trypsin.

Trypsin activates the other proteolytic enzymes, including its proenzyme trypsinogen. The acinar cells also secretes inhibitors which prevents activation of trypsinogen within the pancreas. A failure to

express a normal trypsinogen inhibitor, pancreatic secretory trypsin inhibitor (PSTI),also known as serine protease inhibitor Kazal type 1 (SPINK1),is a cause of familial pancreatitis.

Acinus is a spherical unit which contains 40 acinar cells .Chloride secretion varies inversely with bicarbonate secretion such that the sum of these two remains constant. Bicarbonate secretion, buffers acidic fluid which drains from the stomach into the duodenum . CCK potentiates secretin-stimulated bicarbonate secretion.

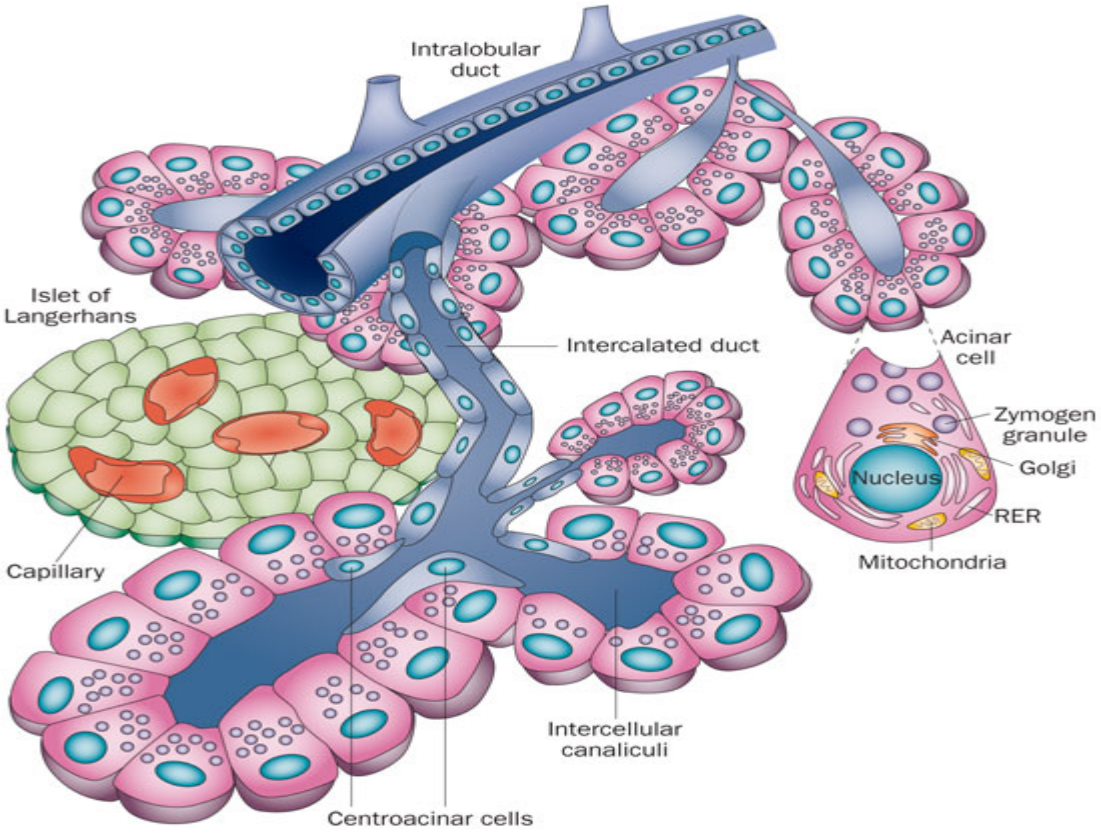


Fig.9 Cellular structure of pancreas

Table.1.PANCREATIC ENZYMES

ENZYME	SUBSTRATE	PRODUCT
Carbohydrate Amylase (active)	Starch, glycogen	Glucose, maltose, maltotriose, Dextrins
Protein Endopeptidases Trypsinogen (inactive) → Trypsin (active) Chymotrypsinogen (inactive) → Chymotrypsin (active) Proelastase (inactive) → Elastase (active) Exopeptidases Procarboxy peptidase A&B (inactive) → Carboxypeptidase A&B (active)	Cleave bonds between amino acids Cleave amino acids from end of peptide chains	Amino acids, dipeptides
Fat Pancreatic lipase (active) Phospholipase A2 (inactive) → Phospholipase A2 (active) Cholesterol esterase	Triglycerides Phospholipase Neutral lipids	2-monoglycerides, fatty acids

2.**Endocrine Pancreas:**

The adult pancreas contains 1 million islets of Langerhans. Most islets contain 3000 to 4000 cells of five major types. Their properties are shown in the table (Table . 2)

Table 2. PANCREATIC ISLET PEPTIDE PRODUCTS

HORMONES	ISLET CELL	FUNCTIONS
Insulin	beta cell	Decreased gluconeogenesis, glycogenolysis, fatty acid breakdown, and ketogenesis Increased glycogenesis, protein synthesis, and glucose uptake
Glucagon	alpha cell	Opposite effects of insulin; increased hepatic glycogenolysis and gluconeogenesis
Somatostatin	delta cell	Inhibits GI secretion Inhibits secretion and action of all GI endocrine peptides Inhibits cell growth
Pancreatic polypeptide	PP cell	Inhibits pancreatic exocrine secretion and section of insulin Facilitates hepatic effect of insulin
Amylin (IAPP) -islet amyloid polypeptide	beta cell	Counterregulates insulin secretion and function
Pancreastatin	beta cell	Decreases insulin and somatostatin release Increases glucagon release Decreases pancreatic exocrine secretion
Ghrelin	epsilon cell	Decreases insulin release and insulin action

PANCREATITIS

Pancreatitis is the acute inflammation of the pancreas.

Pancreatic damage occurs when the digestive enzymes are activated before they are released into the small intestine and begin attacking the pancreas. Pancreatitis is divided into 1.Acute pancreatitis

2.Chronic pancreatitis

ACUTE PANCREATITIS (AP)

Sudden inflammation of the normally existing pancreas is called acute pancreatitis.

Acute pancreatitis may be first attack or relapsing attack with normal gland in between.

CLASSIFICATION OF ACUTE PANCREATITIS

ATLANTA CRITERIA (1992)

- Mild acute pancreatitis (80% cases)

  Acute interstitial /edematous pancreatitis

  - Acute absence of organ failure
  - Absence of local complications

- Severe acute pancreatitis (20%)

  Acute haemorrhagic necrotising(fulminant) pancreatitis

  - Local complication +/-
  - Organ failure defined as

- SBP < 90 mmHg
- PaO2 ≤ 60 mmHg
- GI bleed ≥ 500ml/24hours
- Creatinine ≥ 2 mg/dl after rehydration

-Ranson score ≥ 3 or APACHE ≥ 8

REVISED ATLANTA CRITERIA (2012)

Mild acute pancreatitis

- Absence of organ failure.

- Absence of local complications.

Moderately severe acute pancreatitis

- Local complications +/-

- Transient organ failure (< 48 hours)

Severe acute pancreatitis

-Persistent organ failure (>48 hours) and /or death.

Acute pancreatitis is usually a mild disease with minimal organ dysfunction. However,15-20% of all cases demonstrate severe acute pancreatitis

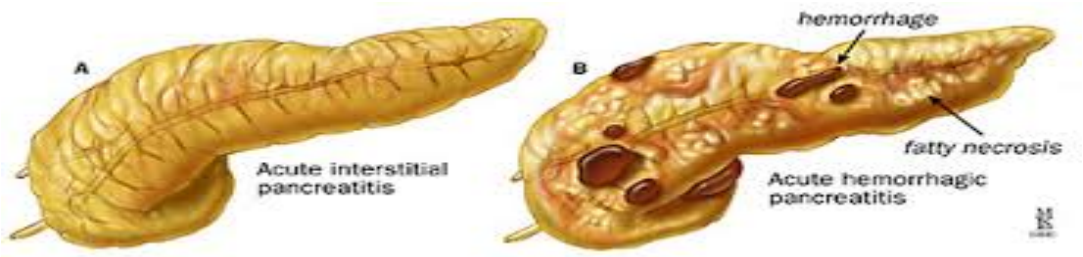


Fig.10.Acute pancreatitis

CHRONIC PANCREATITIS

Chronic pancreatitis is progressive irreversible damage of the pancreas due to chronic inflammation.

It can be 1. Chronic relapsing pancreatitis

2. Chronic persistant pancreatitis with or without calcification in the duct or in the parenchyma

INCIDENCE

Acute pancreatitis is the most common gastrointestinal discharge diagnosis in the United States (274,119 patients in 2009), an incidence which has increased 30% since 2000, and is associated with the highest aggregate inpatient costs at 2.6 billion dollars per year. The crude mortality rate of 1.0/100,000 ranks it as the 14th most fatal illness overall and the ninth most common non cancer gastrointestinal death. The highest incidence recorded in Finland and United States. The racial incidence of acute pancreatitis also shows significant variation related to the prevalence of etiological factors and ethnicity.

The annual incidence of acute pancreatitis in Native Americans is 4 per 100,000 population; in whites it is 5.7 per 100,000 population;

and in blacks it is 20.7 per 100,000 population. Smoking is an independent risk factor for acute pancreatitis. Male preponderance of this

disease is more because of alcoholic etiology. Women also suffer from acute pancreatitis as gallstones occur in higher frequency.

AETIOLOGY AND PATHOLOGY OF ACUTE PANCREATITIS

1. Gall stone- common cause
2. Alcohol
3. External trauma
4. After ERCP,biliary, gastric and splenic surgery.
5. Drugs-Corticosteroids such as prednisolone, diuretics (thiazides), HIV drugs such as didanosine and pentamidine, Anticonvulsant (Valproic acid), Chemotherapeutic drugs such as azathioprine and L-asparaginase, estrogen, antihyperglycemic agents like metformin, vildagliptin and sitagliptin.
6. Infections-Viral-Mumps,Coxsackie,Hepatitis B Bacteria, Legionella, Leptospira, Mycoplasma, Salmonella Fungi-Aspergillus Parasites- Ascaris, Clonorchis sinensis, Cryptosporidium, Toxoplasma.
7. Autoimmune disease
8. Hypercalcemia states arising from hyperparathyroidism, Hyperlipidemia
9. Diabetes.

10. Pancreatic duct obstruction by neoplasm, pancreas divisum, ampullary and duodenal lesions. Less common causes include pancreatic duct stones, vasculitis of small vessels of pancreas, porphyria, scorpion sting(sting of the Trinidadian scorpion Tityus ntrinitatis which causes neurotransmitter discharge from cholinergic nerve terminals, leading to massive production of pancreatic juice, idiopathic.

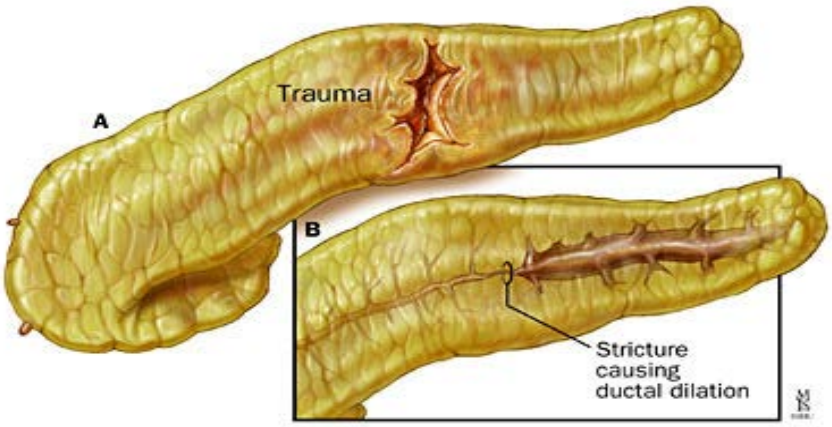


Fig.11.A,B Traumatic pancreatic injury.

PATHOLOGY OF ACUTE PANCREATITIS

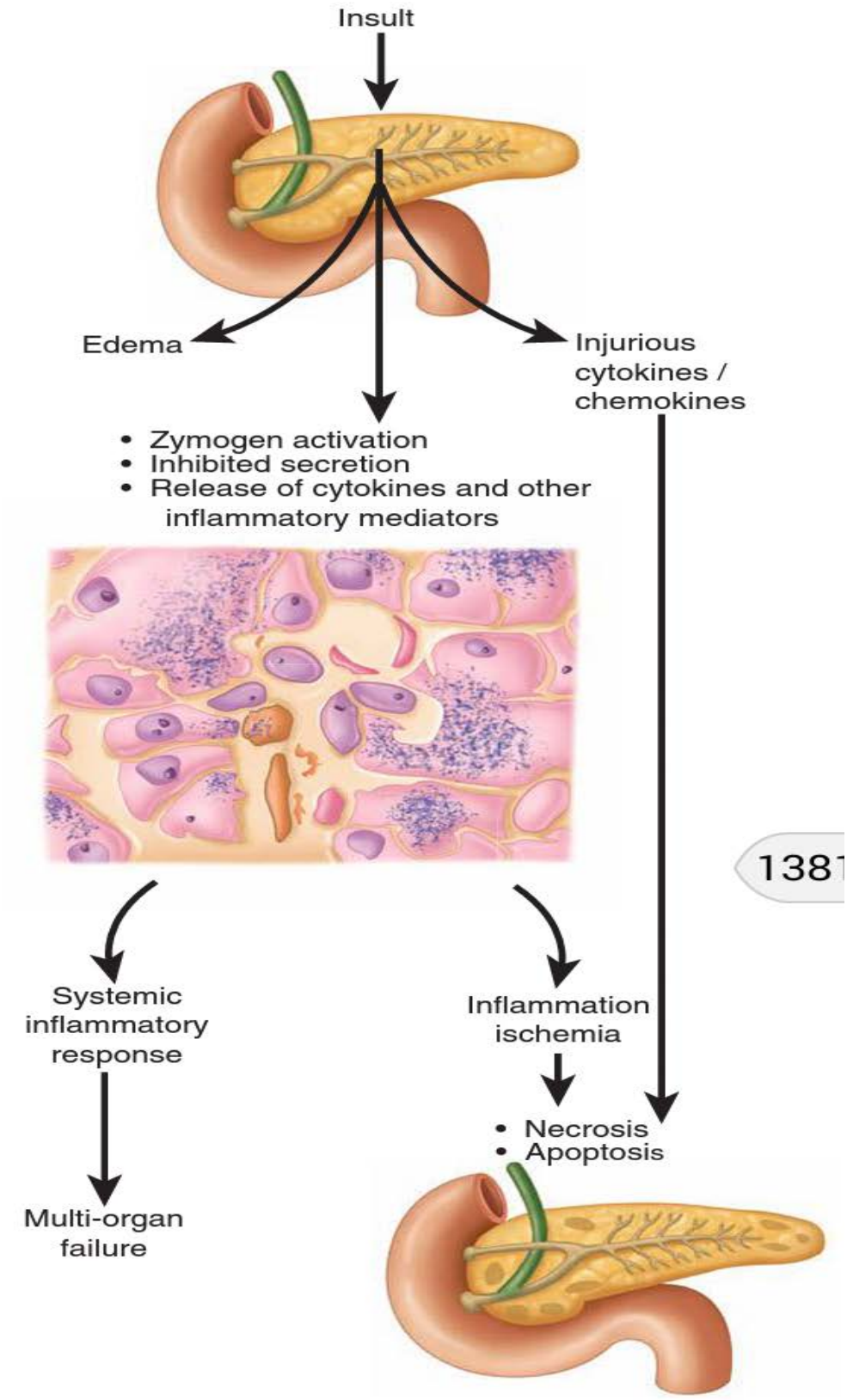


Fig.12.Pathogenesis of acute pancreatitis

GALLSTONES

The main pathology of acute pancreatitis caused by the gallstone disease and other conditions is the obstructed pancreatic duct by gall stones causing ductal hypertension. This ductal hypertension cause the smaller ductules to break and leak the pancreatic secretions into the substance of the pancreas.

In response to ductal obstruction, proteolytic enzymes are activated in the pancreas. The endogenous pancreatic trypsin inhibitor counter small amounts of trypsin. However, destruction of acinar cells occurs due to large amounts of trypsin release and activation of other enzymes.

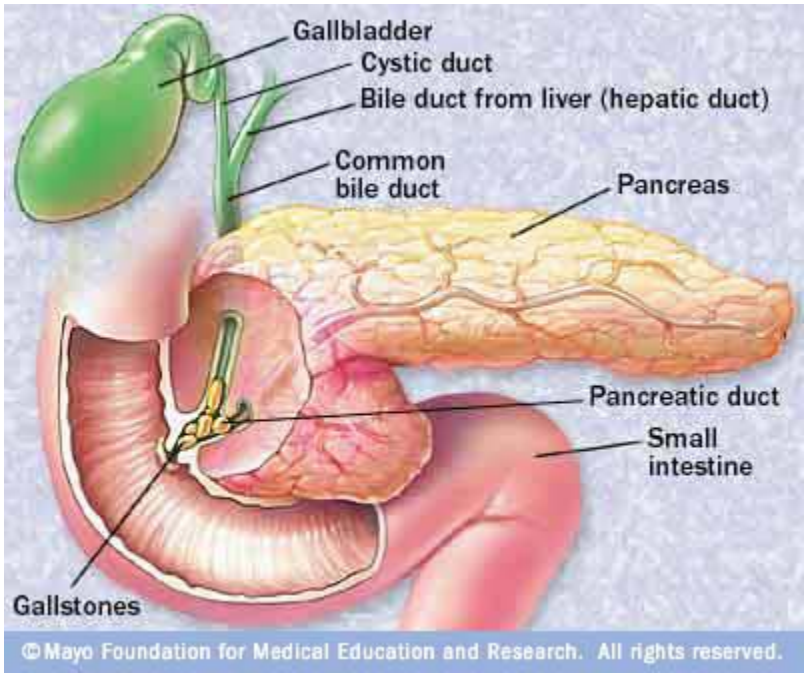


Fig.13.Gallstones in pancreatic duct

INTRAPANCREATIC EVENTS

Focus of tissue injury attract the activated neutrophils and after activation, release superoxides (the “respiratory burst”) and proteolytic enzymes (cathepsins, elastase and collagenase) which cause further injury. The activation of digestive enzyme causes rupture of capillaries and release of amylase, lipase, lecithinase, lysolecithinase, prostaglandins, bradykinins, kallikrein, free radicals, interleukin (IL-6, and IL-8), tumour necrosis factor- alpha, platelet activation factor.

These inflammatory mediators cause the vascular permeability of the pancreas to increase, leading to edema, fluid collection, hemorrhage, and microthrombi. This results in pancreatic hypoperfusion and necrosis, a feature of more severe acute pancreatitis. The interstitital edematous pancreatitis is the acute inflammation of the pancreatic parenchyma and peripancreatic tissues without necrosis.

Necrotizing pancreatitis is acute inflammation of pancreas with necrosis evidenced by pancreatic hypoperfusion with contrast CT.

The updated morphological definitions and the contrast enhanced CT criteria for the diagnosis of the local complications of acute pancreatitis have recently been published in the revised Atlanta statement.

Phospolipase A2 activation causes pulmonary surfactant destruction resulting in liberation of leucotrienes and ARDS.

This is the cause for systemic inflammatory response syndrome (SIRS) resulting in multi organ failure (MOF). When gallstones and other etiological factors cannot be identified there is still the possibility of finding microlithiasis, seen as birefringent crystals, on bile microscopy.30

ALCOHOLIC PANCREATITIS

The amount of alcohol consumed (typically 100 to 150 grams per day) and the pattern of drinking is important rather than the type of alcohol for the cause of acute pancreatitis. Ethanol,which is the metabolic toxin causes a brief secretory increase followed by inhibition, spasm of the sphincter of Oddi, ductal permeability resulting in acute pancreatitis. Ethanol also increases the protein content of pancreatic juice, decreases bicarbonate levels, and trypsin inhibitor concentration.

IATROGENIC

5% to 10% of ERCP causes acute pancreatitis as a complication and it is the third most common identified etiological factor. Surgeries resulting in acute pancreatitis are pancreatic biopsy, exploration of the extrahepatic biliary tree and ampulla of Vater, distal gastrectomy,

splenectomy, colectomy, nephrectomy, aortic aneurysmorraphy, and retroperitoneal lymphadenectomy.

PANCREATITIS DUE TO FAMILIAL ETIOLOGY

It is an autosomal dominant disorder due to trypsinogen gene mutation (PRSS1),and SPINK1 protein mutation.

HYPERLIPIDEMIA

Lipase is thought leading to microcirculatory impairment and ischemia,mainly in patients with types I and V hyperlipoproteinemia associated with hypertriglyceridemia to liberate toxic fatty acids into the pancreatic microcirculation,.

DIAGNOSIS

The first step to make the diagnosis is eliciting the detailed history and careful physical examination. If the patient has a history of colicky pain and binge alcohol consumption,acute pancreatitis should be suspected.

CLINICAL FEATURES:

SYMPTOMS:

- Abdominal pain

  Sudden onset of epigastric pain which is severe constant in nature radiating to the back, relieves by leaning forward

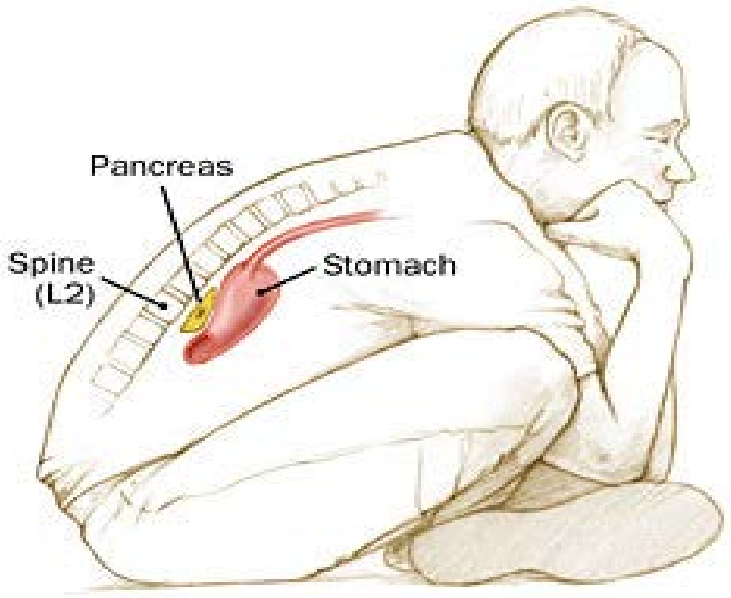


Fig.14.Typical posture for pain relief

- Nausea
- Vomiting
- Anorexia
- Diarrhoea
- Loss of appetite

SIGNS:

- Fever or Chills
- Tachycardia
- Hypotension
- Abdominal tenderness
- Muscular guarding in upper abdomen
- Abdominal distension
- Diminished or absent bowel sounds because of ileus
- Jaundice in few cases
- Dyspnoea due to irritation of diaphragm, pleural effusion (left side) and ARDS
- Severe cases will develops hemodynamic instability, hematemesis, malena, pallor. Muscular spasm in the extremities may be noted secondary to hypocalcemia

Physical findings of severe necrotizing pancreatitis

- Cullen's sign – bluish discolouration around the umbilicus due to hemoperitoneum

Fig.15. Cullen's sign

- Grey-Turner sign-reddish brown discolouration along the flanks due to retroperitoneal blood dissecting along tissue planes

Fig.16. Grey-Turner sign

Fox sign - Ecchymosis in the inguinal region

Erythematous skin nodules located on extensor skin surfaces results from focal subcutaneous fat necrosis.

INVESTIGATIONS

LABORATORY TESTS

- Routine blood investigations-Complete blood count, ESR, blood urea, serum creatinine, serum calcium and coagulation profile.
- **Serum amylase** (Normal value-30-180IU/L)

The level increases immediately with the disease onset and reaches its peak level within several hours. Severity of acute pancreatitis doesn't correlates with raise of serum amylase level. Conditions causing hyperamylasemias other than acute pancreatitis are obstruction of small bowel,duodenal ulcer perforation, or other inflammatory conditions of the abdomen. Urinary amylase levels usually remain elevated for several days after serum levels have returned to normal.

- **Serum lipase** which is more specific for pancreatitis remain raised for longer as it has a longer half life than amylase.
- **Liver function tests**:

AST (Aspartate aminotransferase), ALT (alanine aminotransferase), GGT(gamma-glutamyl Transpeptidase), serum bilirubin and alkaline phosphatase are the tests to differentiate the biliary etiology from other causes of pancreatitis.

- A variety of **single serum parameters**, such as **C-Reactive**

Protein (CRP), alpha1-antitrypsin, polymorphonuclear (PMN) elastase,phospholipaseA2, beta2-macroglobulin, trypsinogen activation peptide procalcitonin, tissue necrosis factor alpha and interleukin 6 and 8 have been investigated and found to be useful markers of the severity of acute pancreatitis. Among these biochemical indicators, the most widely and the simplest available test is CRP.

- C-Reactive Protein (CRP)

CRP is a ring shaped acute-phase protein produced by the liver and found in plasma which is raised as a result of macrophages and T cells activation causing interleukin-6 secretion in inflammation.

CRP is a substance that first identified in patients serum with acute inflammation. It is named as it has reacted with Pneumococcus somatic 'C' carbohydrate antigen.

In 1930, Tillett and Francis discovered CRP. The first chromosome (1q21-q23) is the location of the CRP gene. Upon activation of complement system, by binding of CRP to the phosphocholine which is expressed on the surface of inflammed cells, necrotic and apoptotic cells are cleared by macrophages which promotes phagocytosis. Normal value of CRP concentration in healthy adults is below10 mg/l. CRP raises in inflammation within two hours, and peaks at 48 hours with constant half-life of 18 hours, and therefore CRP is a marker for inflammation and

also predict the severity of the inflammation. Serum CRP levels above 12~15 mg/dl correlate with severe disease.

CRP is a more sensitive than the ESR (Erythrocyte Sedimentation Rate). CRP levels have been evaluated in terms of their ability to predict severity, necrosis and complications of acute pancreatitis. A CRP level of less than or equal to 150mg/dl obtained at 72 hours is useful enough to rule out with high degree of probability the presence of necrosis in acute pancreatitis.

- ❖ Alpha 1 Antitrypsin(AIAT)

Alpha 1 Antitrypsin is a serum trypsin inhibitor belongs to serpin family synthesized by the liver. It protects the tissues from the action of neutrophil- protease enzymes of the inflammatory cells. Normal value of AIAT concentration in healthy adults ranges from 1.5 to 3g/l. Its level rises manyfold upon acute inflammation. Its gene is present in long arm of 14 chromosome (14q321). A1AT deficiency results in respiratory diseases like emphysema, COPD in adults and cirrhosis in adults or children due to break down of elastin by neutrophil elastase. Methods available to detect are enzyme linked immuno sorbent assays, radial immunodiffusion, turbidimetry, nephlometry.

RADIOLOGIC IMAGING

❖ **Plain X – ray chest PA View**

Left sided diaphragm elevation,

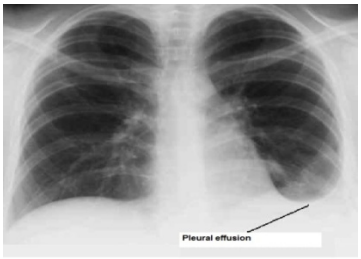
Left sided Pleural effusion

❖ **Plain X-Ray Abdomen**

Mostly non specific findings. Air will be seen in duodenal C-loop.

Sentinel loop sign which is produced by focal dilatation of proximal jejunum. X-ray also shows **Colon cut off sign** which is due to dilatation of colon upto transverse colon with less gas distal to splenic flexure.

Fig.17 X-Rays of acute pancreatitis



Renal halo sign

- In suspected biliary pancreatitis, **Ultrasound** of abdomen is the investigation of choice for confirming the presence of gallstones. It also identifies the extrapancreatic dilatation of the ducts, oedematous pancreas and peripancreatic collection of the fluid.

Fig. 18. Ultrasound of acute pancreatitis

- **CT Scan (Computed Tomography):** CT is the investigation of choice next to ultrasound in acute pancreatitis. It detects the edema of the pancreas, collection of the fluid, and it allows for the grading of severity of acute pancreatitis. It also identifies the complications like pseudocysts, necrosis, infected necrosis and hemorrhage.

CT shows both pancreatic and peripancreatic findings. Pancreatic findings are focal or diffuse enlargement of pancreas, pancreatic edema and necrosis. Peri pancreatic findings are thickening or blurring of adjacent tissues. The CT findings correlate well with the course and severity of the disease.

Plain CT helps in diagnosis of the disease and reveals fluid collection but cannot identify the necrosis or vascular insult. Contrast enhanced CT helps in detecting all complications such as fluid collection, pseudocyst, abscess, pseudoaneurysm. Infected necrosis or abscess is indicated by air bubbles in CE-CT. This scan can also helps for diagnostic fine needle aspiration or placement of pigtail catheter.

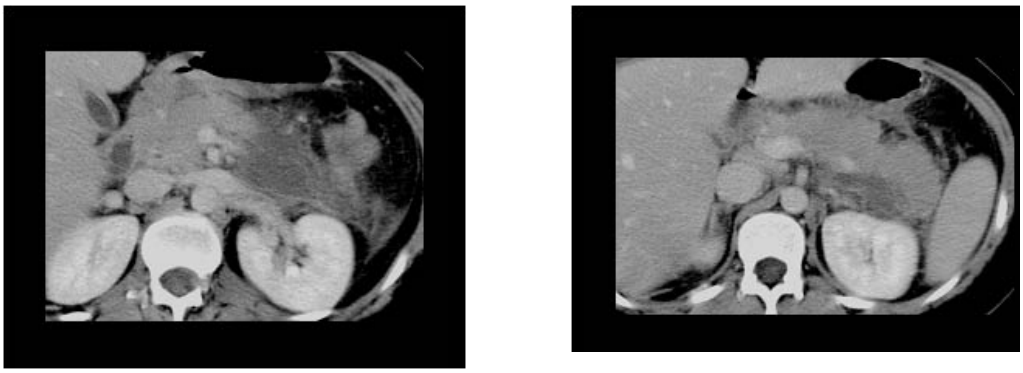


Fig. 19.CT scan of acute biliary pancreatitis

- Magnetic Resonance Cholangiopancreatography (MRCP): MRCP predict the severity of pancreatitis as same as CE- CT. It also identifies the necrosis of the pancreas, but for the detection of small stone it is less sensitive.

Fig.20. Sigmoid configuration of main pancreatic duct with obstruction of papilla.

❖ Endoscopic Ultrasonography: It is the better investigation for obese patients. It also helps to identify the patient for whom therapeutic ERCP would be beneficial.

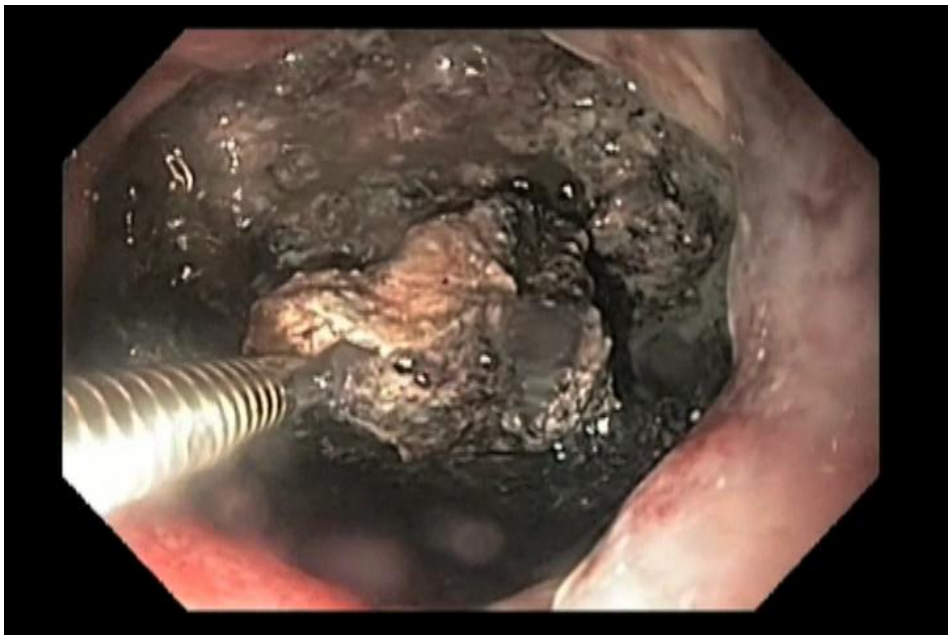


Fig.21.ERCP

SCORING SYSTEMS

For proper decisions of management, it is important to predict the severity of acute pancreatitis. Decisions of management depends on whether a patient should be admitted to an intensive care unit, or transferred to a tertiary hospital, and whether an ERCP is needed, or about necessity of fluid therapy, and other issues. The most widely used prognostic criteria is the Ranson's criteria (Table 3).

This score uses clinical and biochemical parameters in the first 48 hours of admission. The disease is "predicted severe", when there are 3 or more positive criteria. Other scoring systems for predicting severity are Bedside Index for Severity of Acute Pancreatitis (BISAP),an APACHE II score of 8 or more at 24 hours after admission, CT severity index (CTSI),SOFA score, a serum C-Reactive Protein level of >150mg/dl also predict the severity with similar accuracy as Ranson's criteria.

Ranson identified 11 objective parameters, out of these, five are measured at the time of admission,the remaining six are measured within 48 hours of admission.

Table - 3

Ranson's prognostic signs of pancreatitis	
Criteria for acute pancreatitis not due to gallstones	
At admission	During the initial 48 h
Age >55 y	Hematocrit fall >10 points
WBC >16,000/mm^3	BUN elevation >5 mg/dL
Blood glucose >200 mg/dL	Serum calcium <8 mg/dL
Serum LDH >350 IU/L	Arterial PO_2 <60 mm Hg
Serum AST >250 U/dL	Base deficit >4 mEq/L
	Estimated fluid sequestration >6 L
Criteria for acute gallstone pancreatitis	
At admission	During the initial 48 h
Age >70 y	Hematocrit fall >10 points
WBC >18,000/mm^3	BUN elevation >2 mg/dL
Blood glucose >220 mg/dL	Serum calcium <8 mg/dL
Serum LDH >400 IU/L	Base deficit >5 mEq/L
Serum AST >250 U/dL	Estimated fluid sequestration >4 L

AST = aspartate transaminase; BUN = blood urea nitrogen; LDH = lactate dehydrogen-ase; PO_2 = partial pressure of oxygen; WBC = white blood cell count.
Note: Fewer than 3 positive criteria predict mild, uncomplicated disease whereas more than 6 positive criteria predict severe disease with a mortality risk of 50%.

Table – 4 Glasgow Imrie criteria for acute pancreatitis 3 or more of the below in first 48 hours indicates a severe attack.

Pao2	< 8 KPa
Age	>55 years
Neutrophils	>15x10^9/L
Calcium	>2 mmol/L
Renal function	Urea>16mmol/L
Enzymes	LDH>600 IU/L, AST>2000 IU/L
Albumin	<32g/L
Sugar	Glucose>10mmol/L

Table – 5 BISAP SCORE

- BUN > 25
- Impaired mental status
- SIRS (> 2 criteria)
- Age > 60 yrs
- Pleural effusion on CT scan
- 1 point for the presence of each finding.

BUN, blood urea nitrogen; SIRS, systemic inflammatory response syndrome

BISAP Score	Observed Mortlity
0	0.1%
1	0.4%
2	1.6%
3	3.6%
4	7.4%
5	9.5%

Table - 6

The acute physiology and chronic health evaluation (APACHE-II) score

A.ACUTE PHYSIOLOGY SCORE

Physiologic Variable	High Abnormal Range					Low Abnormal Range				
	+4	+3	+2	+1	0	+1	+2	+3	+4	Points
Temperature - rectal (°C)	≥41°	39 to 40.9°		38.5 to 38.9°	36 to 38.4°	34 to 35.9°	32 to 33.9°	30 to 31.9°	≤29.9°	
Mean Arterial Pressure - mm Hg	≥160	130 to 159	110 to 129		70 to 109		50 to 69		≤49	
Heart Rate (ventricular response)	≥180	140 to 179	110 to 139		70 to 109		55 to 69	40 to 54	≤39	
Respiratory Rate (non-ventilated or ventilated)	≥50	35 to 49		25 to 34	12 to 24	10 to 11	6 to 9		≤5	
Oxygenation: A-aDO2 **or** PaO2 (mm Hg) a. FIO2 ≥0.5 record A-aDO2 b. FIO2 <0.5 record PaO2	≥500	350 to 499	200 to 349		<200 PO2>70	 PO2 61 to 70		 PO2 55 to 60	 PO2<55	
Arterial pH (preferred) Serum HCO3 (venous mEq/l) (not preferred, but may use if no ABGs)	≥7.7 ≥52	7.6 to 7.69 41 to 51.9		7.5 to 7.59 32 to 40.9	7.33 to 7.49 22 to 31.9		7.25 to 7.32 18 to 21.9	7.15 to 7.24 15 to 17.9	<7.15 <15	
Serum Sodium (mEq/l)	≥180	160 to 179	155 to 159	150 to 154	130 to 149		120 to 129	111 to 119	≤110	
Serum Potassium (mEq/l)	≥7	6 to 6.9		5.5 to 5.9	3.5 to 5.4	3 to 3.4	2.5 to 2.9		<2.5	
Serum Creatinine (mg/dl) Double point score for acute renal failure	≥3.5	2 to 3.4	1.5 to 1.9		0.6 to 1.4		<0.6			
Hematocrit (%)	≥60		50 to 59.9	46 to 49.9	30 to 45.9		20 to 29.9		<20	
White Blood Count (total/mm3) (in 1000s)	≥40		20 to 39.9	15 to 19.9	3 to 14.9		1 to 2.9		<1	
Glasgow Coma Score (GCS) Score = 15 minus actual GCS										
A. Total Acute Physiology Score (sum of 12 above points)										
B. Age points (years) ≤44=0; 45 to 54=2; 55 to 64=3; 65 to 74=5; ≥75=6										
C. Chronic Health Points (see below)										
Total APACHE II Score (add together the points from A+B+C)										

Total acute physiology score(sum of 12 above points)

Table – 7

C.CHRONIC HEALTH POINTS IN APACHE II

HISTORY	POINTS FOR ELECTIVE SURGERY	POINTS FOR EMERGENCY SURGERY
Proven liver cirrhosis,portal hypertension or liver failure	2	5
Cardiovascular NYHA grade IV	2	5
Respiratory eg:COPD, pulmonary hypertension	2	5
Chronic renal disease	2	5
Immunocompromised	2	5

Total APACHE II Score is the sum of points from A+B+C

Score 0 to 2 : 2 % mortality

Score 3 to 4 : 15% mortality

Score 5 to 6 : 40% mortality

Score 7 to 8 : 100% mortality

In 1991, **APACHE III SCORE** was released with the aim to improve statistical power, predict the patient outcome and to identify factors in ICU which influence outcome variations.

Parameters includes 17 physiological variables and Total score (0-299), acid base disturbances, GCS Score-based on the worst, Age score, 7 Co-morbidities (cardiac, respiratory and renal failures excluded).

In 1985, Balthazar introduced **CT severity index** for determining the morphologic severity of acute pancreatitis.

Table-8. Scoring for pancreatic necrosis

0 point	No pancreatic necrosis
2 points	≤30% pancreatic necrosis
4 points	>30%-50% pancreatic necrosis
6 points	>50% pancreatic necrosis

Table-9. Modified CT severity index Evaluation of pancreatic morphology, without taking into account the extent of pancreatic necrosis

0 points, Grade A	Normal pancreas consistent with mild pancreatitis
2 points, Grade B/C	Focal or diffuse enlargement of the gland including contour irregularities and inhomogeneous attenuation with or without peripancreatic inflammation.
4 points, Grade D/E	Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis
Additional 2 points	Extra-pancreatic complications (one or more of the following: pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)

CT SEVERITY SCORE:

Score 0- : 3% mortality

Score 4-6 : 6% mortality

Score 7-10 : 17% mortality

Organ failure is scored using the Marshall or Sequential Organ Failure Assessment (**SOFA)** systems.

Multiple organ failure is defined as two or more organs registering 2 or more points on these scoring systems. The three organ systems most frequently involved are cardiovascular, respiratory, and renal. Monitoring organ failure over time and in response to treatment is important in the clinical care of the disease.

Table – 10 SOFA-Sequential organ failure assessment score in acute pancreatitis

SOFA-Sequential organ failure assessment score in acute pancreatitis49

	0	1	2	3	4
Respiration (PaO2FIO2) (mm Hg)	>400	<400	<300	<200 with respiratory Support	<100 with respiratory support
Coagulation Platelets (xl01 per µL)	>150	<150	<100	<50	<20
Liver Bilirubin (µmol/L)	<20	20–32	33–101	102–204	>204
Cardiovascular Hypotension	No Hypotension	MAP <70mm Hg	Dopamine <5 or dobutamine (any dose)*	Dopamine >5 or epi <0.1*or norepi <0.1*	Dopamine >15 or epi >0.1*or norepi >0.1*
Central nervous system Glasgow coma score	15	13–14	10–12	6–9	<6
Kidney Creatinine (µmol/L) or urine output	<110	110–170	171–299	300–440 or <500 ml/day	>440 or <200 ml/day

MAP = mean arterial pressure. Epi = epinephrine. Norepi = norepinephrine. *Adrenergic agents administered for at least 1 h (doses given in µg/kg per min).
A score of 2 or more in any two systems indicates the presence of multiple organ failure.

Table-11 .Scoring system to assess severity of acute pancreatitis

MILD TO MODERATE AP	**SEVERE AP**
Ranson signs ≤ 3	Ranson signs > 3
APACHE II score ≤ 8	APACHE II score > 8
CRP < 100 mg/l	CRP >100 mg/l
Balthazar's CT score ≤ 3	Balthazar's CT score > 3

TREATMENT

The patient should be admitted in the hospital**.** The diagnosis should be established with clinical features, laboratory investigations and imaging studies.

Pain must be controlled using NSAIDS for mild patients and opioid analgesics in severe cases.

Fluid therapy should be initiated with ringer lactate, normal saline, dextrose, plasma, fresh blood transfusion/packed cells, as there is lot of fluid sequestration and third space loss . Ringer lactate solution is found to be superior to normal saline in reducing the systemic inflammatory response on the basis of recent data.

Continuous nasogastric aspiration, urinary catheterization should be done for monitoring urine output 50 ml hourly. Enteral nutrition should be started early after fluid therapy within first 24 hours of admission through nasogastric tube.

Antibiotics such as third generation cephalosporins are of no proven value unless otherwise suspected infection.

For preventing stress ulcers iv Pantoprazole 80mg bd should be given.

Frequent examination of the patients and serial measurement of inflammatory markers is the must for identification of early complication.

CECT should be ordered for suspected complicated cases.

For severe cases, intervention should be carried out invasively. The treatment for biliary pancreatitis is cholecystectomy and clearance of common duct by ERCP if stone is present in CBD. If there is persistence of obstruction after 24 hours,it leads to biliary sepsis, so emergency ERCP has to be done.

COMPLICATIONS

Both local and systemic complications can occur in acute pancreatitis.

Table-12 Local complications of acute pancreatitis[39]

CONTENT	Acute (<4 weeks, no defined wall)		Chronic (>4 weeks, defined wall)	
	NO INFECTION	INFECTION	NO INFECTION	INFECTION
FLUID	Acute pancreatic fluid collection (APFC)	Infected APFC	Pseudocyst	Infected pseudocyst
SOLID+/- FLUID	Acute necrotic collection (ANC)	Infected ANC	Walled off necrosis (WON)	Infected WON

Table - 13 Systemic complications

SYSTEM	COMPLICATIONS
1. Cardiovascular	1. Hypotension 2. Hypovolemia 3. Sudden death 4. Nonspecific ST-T wave changes 5. Pericardial effusion
2. Pulmonary	1. Pneumonia, atelectasis 2. Acute respiratory distress syndrome 3. Pleural effusion
3. Renal	1. Oliguria 2. Azotemia 3. Renal artery/vein thrombosis
4. Hematologic	1. Hemoconcentration 2. Disseminated intravascular coagulopathy
5. GI hemorrhage	1. Peptic ulcer 2. Erosive gastritis 3. Portal vein or splenic vein thrombosis with varices 4.Persistant duodenal ileus
6. Metabolic	1. Hyperglycemia 2. Hypocalcemia 3. Hypertriglyceridemia 4. Encephalopathy 5. Sudden blindness (Purtscher's retinopathy)
7. Central nervous system	1. Psychosis 2. Fat emboli 3. Alcohol withdrawal syndrome

Table-14 Four categories of acute pancreatitis severity based on organ failure and local complications

Determinants	No local complications	Sterile local complications	Infected local complications
No organ failure	Mild	Moderate	Severe
Transient organ failure	Moderate	Moderate	Severe
Persistent organ Failure	Severe	Severe	

AIMS AND OBJECTIVES

Primary objective is study on C-Reactive Protein for assessing and monitoring the severity of acute pancreatitis.

METHODS AND MATERIALS

TITLE

Study on C- Reactive Protein-an aid for assessing and monitoring the severity of acute pancreatitis in Government Mohan Kumaramangalam Medical College, Salem

SOURCE OF DATA:

All Patients with acute pancreatitis who admitted in department of general surgery in Government Mohan Kumaramangalam Medical College Hospital.

SAMPLE SIZE:

Sample size of 75 patients fulfilling the inclusion criteria.

METHOD OF COLLECTION OF DATA:

The data was collected by thorough history taking, clinical examination and investigations.

STUDY DESIGN

Prospective study

STUDY PERIOD

June 2015 - June 2016

PLACE OF THE STUDY

Govt. Mohan Kumaramangalam Medical College Hospital, Salem.

ETHICAL CLEARANCE

Ethical clearance obtained from the institution ethical committee

Inclusion criteria:

Patients with acute Pancreatitis admitted in general surgery ward

Exclusion criteria:

1.Patients admitted with acute pancreatitis who are associated with comorbid disease at the time of admission.

2.Patients who come with complications of acute pancreatitis

3.Patients with chronic pancreatitis

STUDY METHODOLOGY

In this study, prior written informal consent was obtained from the patients admitted with acute pancreatitis. Data such as detailed history taking and complete clinical examination was noted on a pretested proforma. All patients were subjected to following investigations.

Complete blood count, Blood sugar, urea,

Serum creatinine, serum electrolytes, Urine routine examination,

Serum Amylase, Serum Lipase

C Reactive Protein, Alpha 1 Antitrypsin,

ECG in all leads, Chest X ray PA view and/or USG Thorax,

Ultrasound Abdomen,

Relevant special investigation: CT Abdomen-Plain/Contrast

- The details of the above were given in the clinical proforma sheet and in the master chart.

The results are evaluated and analyzed by comparing with serial monitoring of alpha 1 antitrypsin. White cell count, erythrocyte sedimentation rate, temperature were used as reference data.

RESULTS

The collected data were analysed with IBM.SPSS statistics software 23.0

Version. To describe about the data descriptive statistics mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t-test was used. The logistic regression model with forward stepwise (Wald) method was used to find the influence of variables in the severity of disease.

In both the above statistical tools the probability value .05 is considered as significant level.

P - Value** Highly Significant at $P \leq .01$

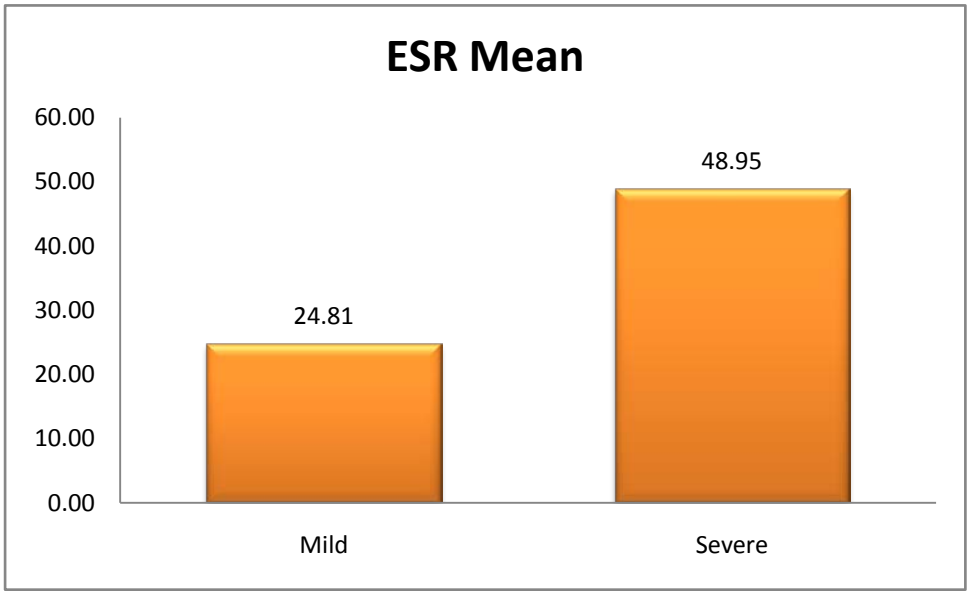


Chart – 1 ESR mean of mild & severe AP

The mean ESR of mild cases was found about 24.81

The mean ESR of severe cases was 48.95 which is more than mild cases and found statistically significant (P<0.001)

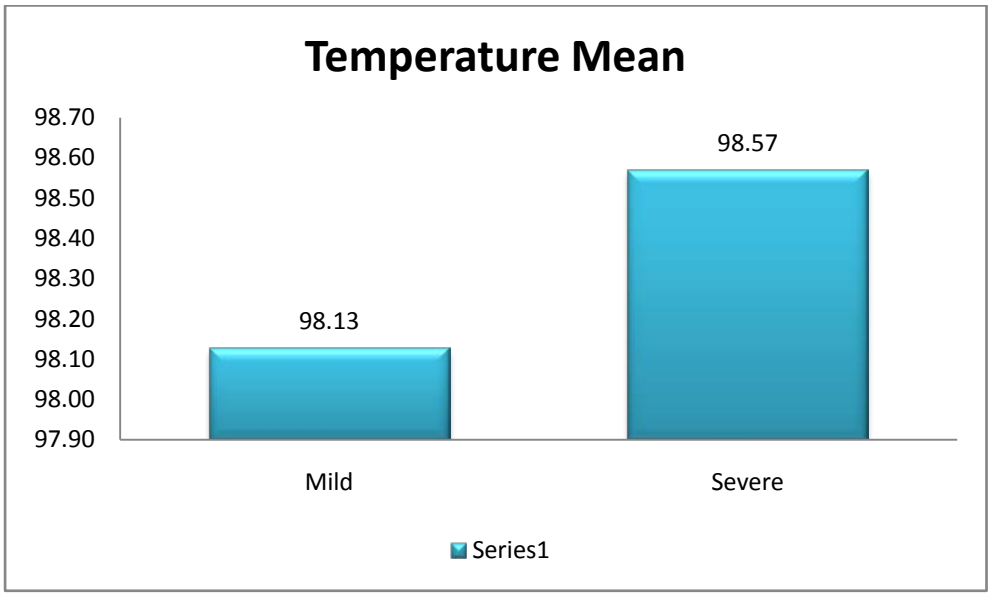


Chart – 2 Temperature mean of mild & severe AP

The mean temperature of mild cases was found about 98.13

The mean temperature of severe cases was about 98.57

It was found statistically significant (P<0.001)

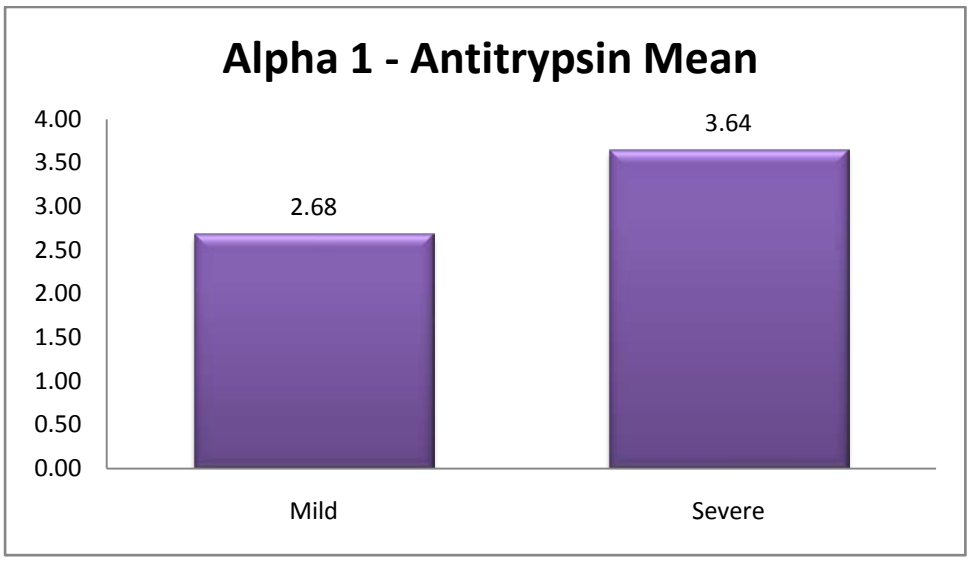


Chart – 3 Alpha 1 Antitrypsin mean of mild & severe AP

The mean Alpha 1 Antitrypsin of mild cases was found about 2.68 and the same for severe cases was about 3.64, which is more than mild cases and was also found statistically significant ($P<0.001$)

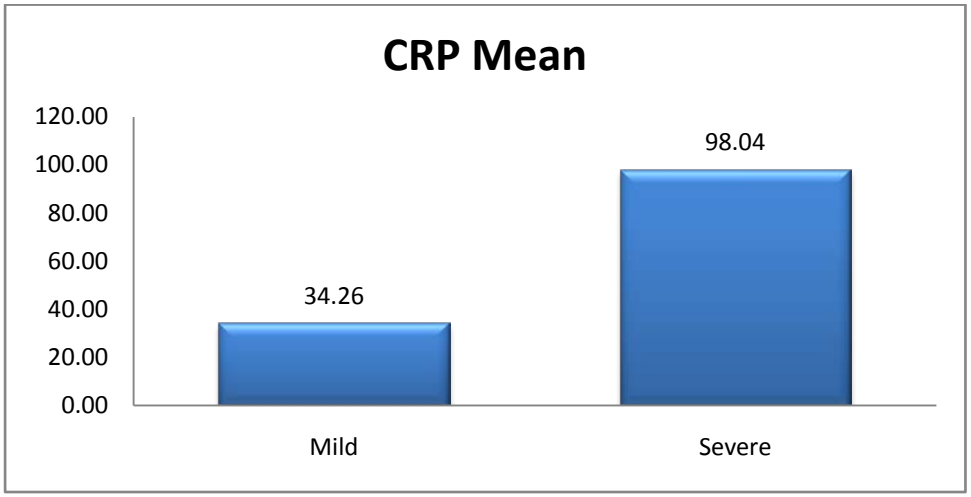


Chart – 4 CRP mean of mild & severe AP

The mean CRP of mild cases was found about 34.26 and the same for severe cases was about 98.04, which is more than mild cases and it was also found statistically significant (P<0.001).

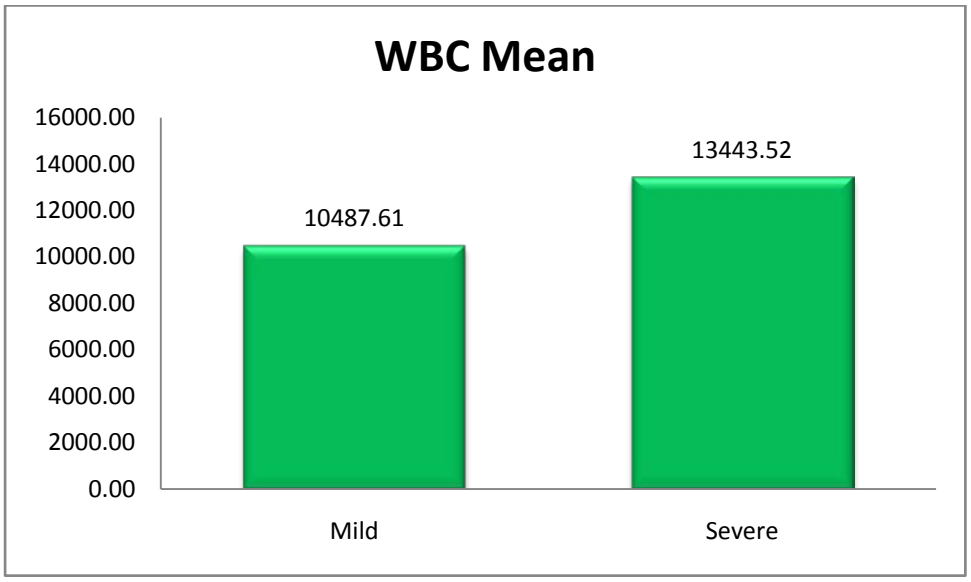


Chart – 5 WBC mean of mild & severe AP

The mean WBC of mild cases was found about 10487.61 and the same for severe cases was about 13443.52 which is more than mild cases and it also found statistically significant (P<0.001).

Table - 15

Group Statistics

Statistics of mean values of inflammatory markers

MS		N	Mean	Std. Deviation	Std. Error Mean
ESR MEAN	Mild	39	24.81	6.71404	1.07511
	Severe	36	48.95	6.51343	1.08557
TEMP MEAN	Mild	39	98.13	.26342	.04218
	Severe	36	98.57	.52459	.08743
AA MEAN	Mild	39	2.6778	.32217	.05159
	Severe	36	3.6432	.56599	.09433
CRP MEAN	Mild	39	34.26	8.42058	1.34837
	Severe	36	98.04	18.84195	3.14033
WBC MEAN	Mild	39	10487.61	1022.1	163.7
	Severe	36	13443.52	691.6	115.3

Table - 16

T – Test Statistics

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	Df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
ESR MEAN	Equal variances assumed	1.311	.256	-15.785	73	.0005	-24.14601	1.52973	-27.19475	-21.09727
	Equal variances not assumed			-15.804	72.812	.000	-24.14601	1.52785	-27.19114	-21.10088
TEMP MEAN	Equal variances assumed	29.522	.000	-4.657	73	.0005	-.44124	.09475	-.63008	-.25240

	Equal variances not assumed			-4.545	50.660	.000	-.44124	.09708	-.63616	-.24632
AA MEAN	Equal variances assumed	2.913	.092	-9.168	73	.0005	-.96546	.10531	-1.17535	-.75558
	Equal variances not assumed			-8.980	54.570	.000	-.96546	.10752	-1.18097	-.74996
CRP MEAN	Equal variances assumed	3.815	.055	-19.175	73	.0005	-63.78063	3.32631	-70.40996	-57.15129
	Equal variances not assumed			-18.663	47.605	.000	-63.78063	3.41757	-70.65358	-56.90768
WBC MEAN	Equal variances assumed	4.570	.036	-14.545	73	.000	-2955.9	203.2	-3360.9	-2550.9
	Equal variances not assumed			-14.766	67.114	.0005	-2955.9	200.2	-3355.5	-2556.4

Day wise ESR mean values of mild and severe cases

	Mild	Severe
Day 1	37.05	71.94
Day 3	31.13	63.50
Day 5	27.69	55.50
Day 7	22.00	44.75
Day 9	17.05	34.28
Day 11	13.92	23.75

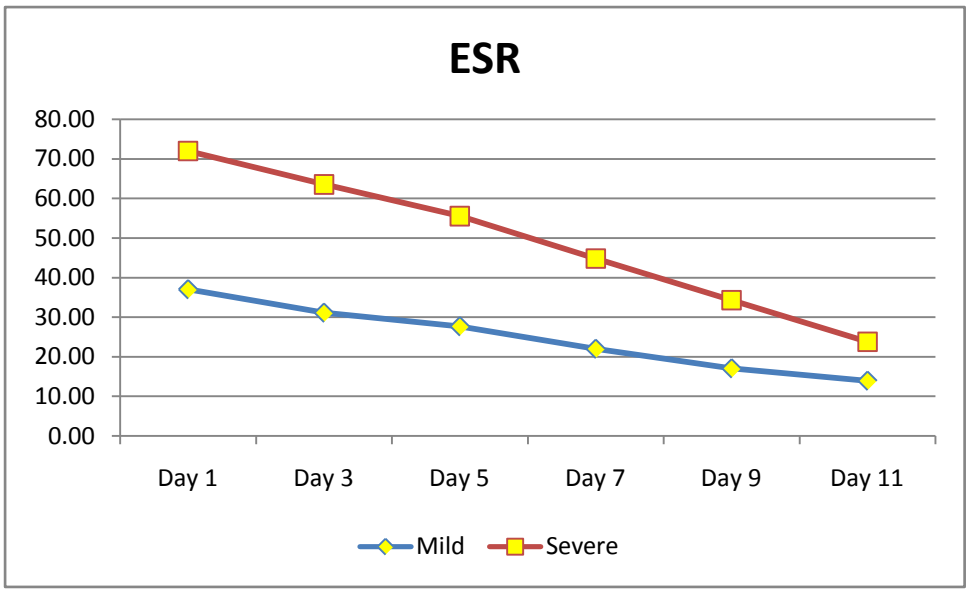


Chart - 6

The mean value of ESR for severe acute pancreatitis on day 1,was 71.94 and the same for mild attack was 37.05, and both were found raised than normal value on day 1.

The mean values of ESR for both severe and mild attacks were declined on days 3,5,7,9,11 and the values were found almost the same by day 11.

Day wise Temperature mean values of mild and severe cases

	Mild	Severe
Day 1	98.26	99.36
Day 3	98.31	99.33
Day 5	98.21	98.64
Day 7	98.00	98.08
Day 9	98.00	98.00
Day 11	98.00	98.00

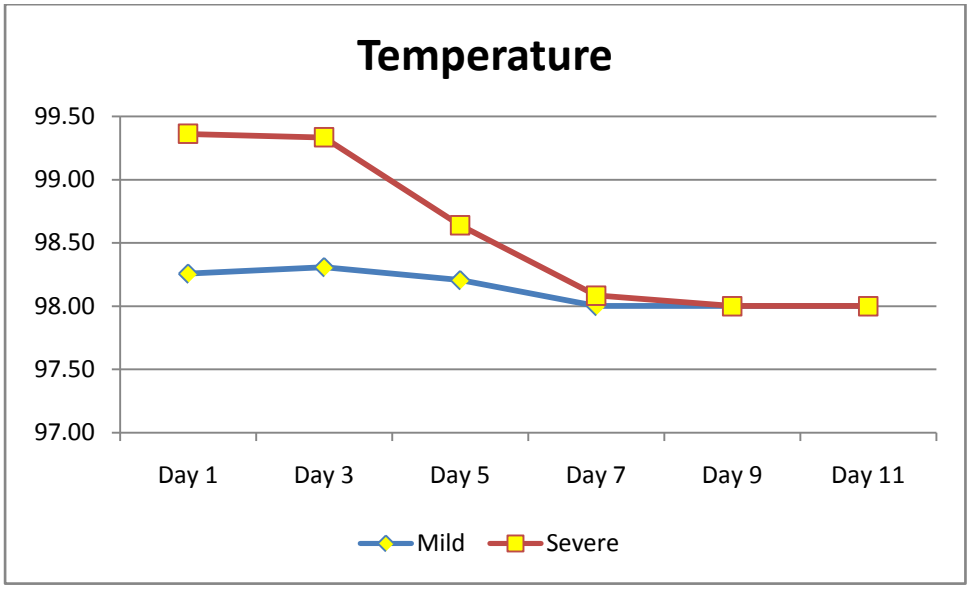


Chart - 7

The mean temperature of severe acute pancreatitis on day 1,was 99.36 and the same for mild attack was 98.26,and both were found raised on day 1. The mean temperatures of both severe and mild attacks declined

on days 3,5,7,9 and the patient was afebrile by day 9 in both groups. Here also, differences were narrow by the end of day 9.

Day wise Alpha 1 Anti-Trypsin mean values of mild and severe cases

	Mild	Severe
Day 1	3.21	4.44
Day 3	3.15	4.22
Day 5	2.96	4.05
Day 7	2.57	3.49
Day 9	2.16	3.00
Day 11	2.02	2.71

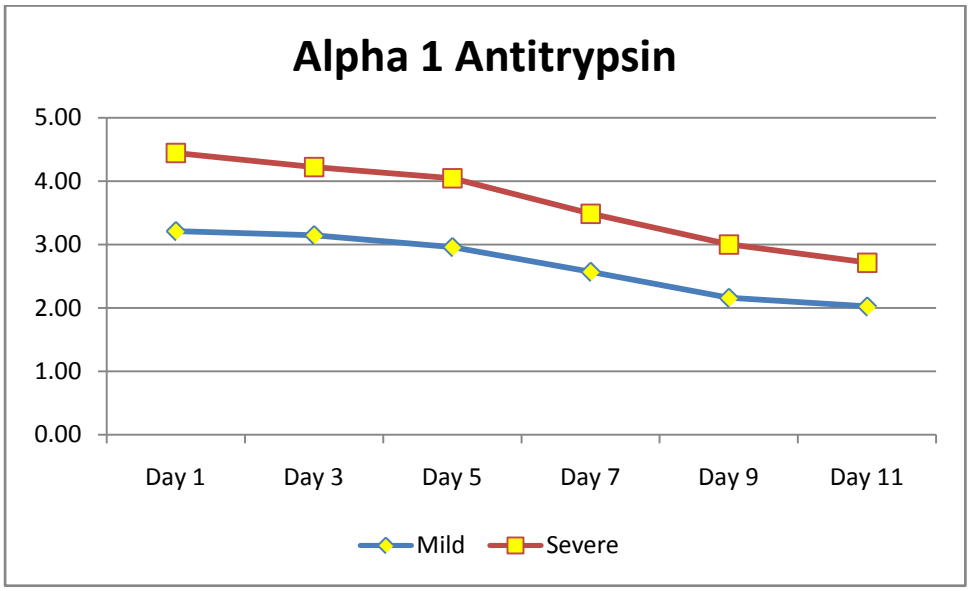


Chart - 8

The mean value of Alpha 1 Antitrypsin for severe acute pancreatitis on day 1,was 4.44 and the same for mild attack was 3.21,and both were found raised on day 1 found statistically significant (P<0.001).

The mean values of Alpha 1 Antitrypsin for both severe and mild attacks were decreased on days 3,5,7,9,11 and the values were almost the same by day 11.

Daywise C-Reactive Protein mean values of mild and severe cases

	Mild	Severe
Day 1	48.97	114.47
Day 3	55.23	137.17
Day 5	44.36	117.58
Day 7	30.62	92.03
Day 9	17.90	69.37
Day 11	8.46	48.60

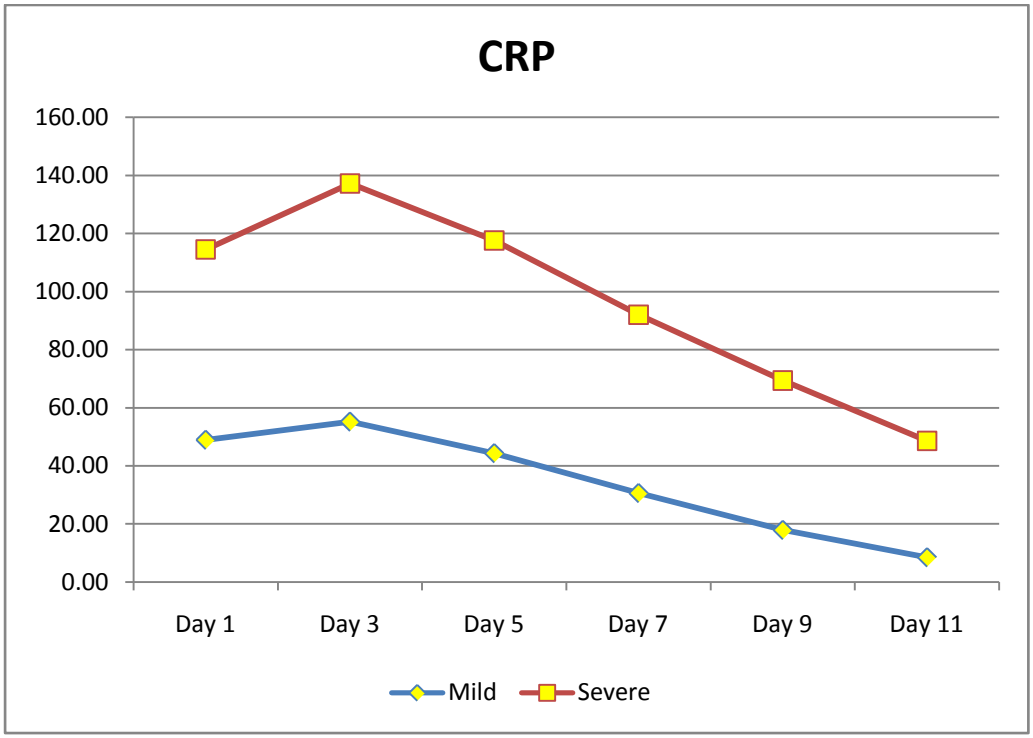


Chart - 9

The CRP mean value for severe acute pancreatitis on day 1,was 114.47 and the same for mild attack was 48.97,and both were found raised on day 1 which was statistically significant($p<0.001$)

The mean values of CRP for both mild and severe acute pancreatitis were found raised on day 3 than the first day, started declining on days 5, 7,9,11. Increase of CRP values was more in patients with severe disease than with mild disease. But the rate of fall of CRP was slow in severe acute pancreatitis and on day 11 difference of values between the two attacks were in a wide range.

Day wise C-Reactive Protein mean values of mild and severe cases

	Mild	Severe
Day 1	10697.44	13936.11
Day 3	10738.46	13911.11
Day 5	10743.59	13877.78
Day 7	10310.26	13077.78
Day 9	10225.64	12936.11
Day 11	10210.26	12922.22

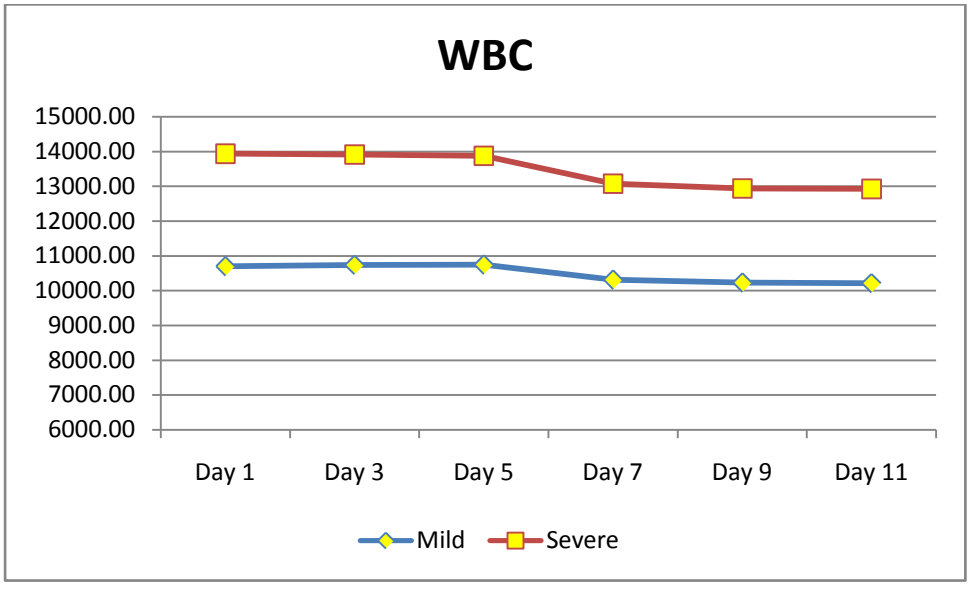


Chart - 10

The WBC mean value for severe acute pancreatitis on day 1, was 13936.11cells per cubic millimeters of blood and the same for mild attack was 10697.44 cells/cu.mm ,and both were found raised on day 1. The values decreased on days 3, 5, 7,9,11. By the end of first week there was narrow range of differences in severe and mild disease.

Logistic Regression

Case Processing Summary

Unweighted Cases[a]		N	Percent
Selected Cases	Included in Analysis	75	100.0
	Missing Cases	0	0.0
	Total	75	100.0
Unselected Cases		0	0.0
Total		75	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
Mild	0
Severe	1

Classification Table

Observed			Predicted		
			Mild	Severe	Percentage Correct
Step 0	MS	Mild	39	0	100.0
		Severe	36	0	0.0
	Overall Percentage				52.0

a. Constant is included in the model.

b. The cut value is .500

Variables in the Equation

	B	S.E.	Wald	df	Sig	Exp(B)
Step 0 Constant	-.080	.231	.120	1	.729	.923

Variables not in the Equation

		Score	df	Sig.
Variables	AGE	.016	1	.899
	ESR MEAN	58.005	1	.000
	TMP MEAN	17.177	1	.000
	AA MEAN	48.706	1	.000
	CRP MEAN	62.576	1	.000
	WBC MEAN	55.759	1	.000
Overall Statistics		67.141	6	.000

Block 1: Method = Forward Stepwise (Wald)

Omnibus Tests of Model Coefficients

	Chi-square	Df	Sig.
Step	103.852	1	.000
Block	103.852	1	.000
Model	103.852	1	.000

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	.000[a]	.750	1.000

a. Estimation terminated at iteration number 20 because maximum iterations has been reached. Final solution cannot be found.

Classification Table

Observed			Predicted		
			MS		Percentage Correct
			Mild	Severe	
Step 1	MS	Mild	39	0	100.0
			0	36	100.0
	Overall Percentage				

a. The cut value is .500

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1	CRP MEAN	7.229	284.254	.001	1	.980	1379.5
	Constant	-480.861	18989.697	.001	1	.980	.0

a. Variable(s) entered on step 1: CRPMEAN.

b. Stepwise procedure stopped because removing the least significant variable result in a previously fitted model.

Variables not in the Equation

		Score	df	Sig.
Variables	AGE	.000	1	.998
	ESR MEAN	.003	1	.960
	TMP MEAN	.171	1	.679
	AA MEAN	.001	1	.975
	WBC MEAN	.006	1	.939

a. Residual Chi-Squares are not computed because of redundancies.

Descriptives

Groups = Mild

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	39	19	65	38.46	9.894
Valid N (listwise)	39				

a. Groups = Mild

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	36	14	65	38.17	10.457
Valid N (listwise)	36				

a. Groups = Severe

DISCUSSION

In our study of 75 patients of acute pancreatitis, 39 patients were found to have mild disease and 36 patients were found to have severe acute pancreatitis according to Atlanta criteria 2012. Etiologies of the disease were alcoholic, biliary and idiopathic.

In 1984, Mayer et al. studied the CRP role in the assessment and monitoring of acute pancreatitis and found that the main value of CRP provided guide to the severity of inflammation. They also found that when the CRP value remain high (> 100 mg/l) at the end of the first week of the illness there is risk of developing pancreatic collections.

In 1984, McMahon et al. found low levels of CRP in patients with mild pancreatitis and high values in patients with severe pancreatitis.

Samples for CRP, Alpha 1 Antitrypsin, WBC, and ESR were collected on day1 of admission and on days 3, 5, 7, 9, 11 after admission. Temperature, ESR, Alpha1Antitrypsin values didn't discriminate acute pancreatitis as mild and severe disease. Although those values were high in severe acute pancreatitis, mean 95% confidence limits of mild and severe attacks were overlapped throughout the study.

On day 1 of admission, difference in WBC count between mild and severe disease, helps to discriminate between the two. As the disease

progressed, CRP values reaches maximum in the end of first week, in severe acute pancreatitis and it takes more time to fall towards normal value. Hence CRP helped to differentiated between mild and severe acute pancreatitis better than WBC and Alpha 1 antitrypsin value. High level of CRP (>100mg /l) at first week suggests that patients who have the disease requires 2 or more weeks to recover and there is risk of developing pancreatic collection.

Increased values of CRP reflect severe local inflammation in mild disease with benign clinical course.

Hence, CRP is a sensitive indicator of continuing inflammation and it may be of better value in selecting the cases who are more prone for developing high risk complications.

We followed the patient,in due course we found the following results

OUTCOME	NUMBER OF PATIENTS
Improvement	39
Worsening of the disease	15
Multiorgan failure	2
Needing surgical intervention for acute Pancreatitis	7
Chronic pancreatitis	20
Death	1

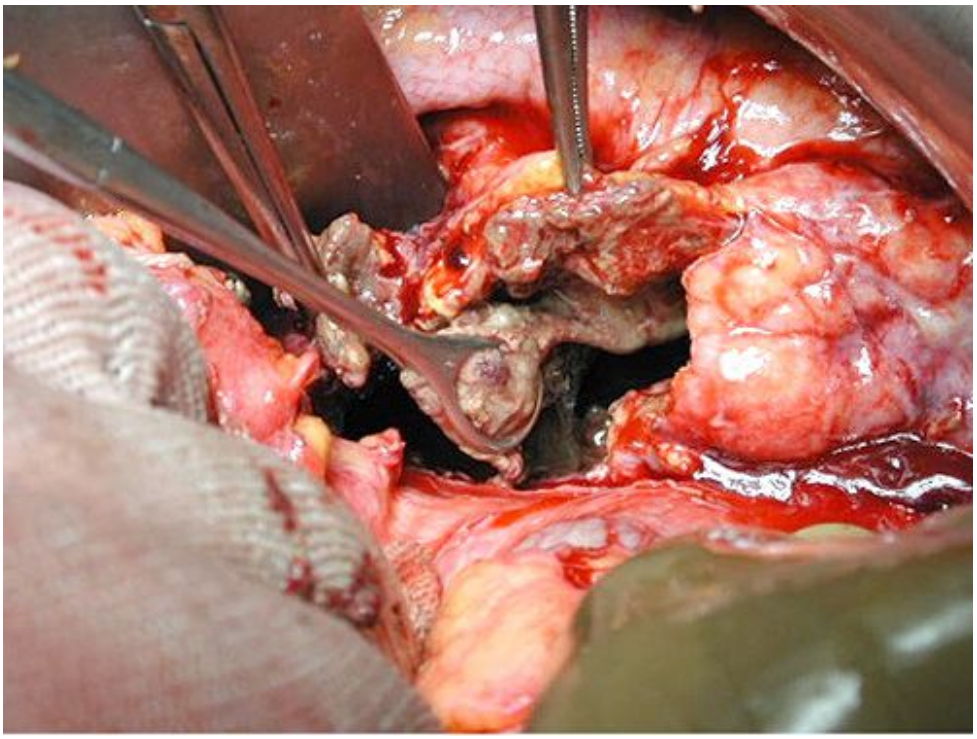
CONCLUSION

Temperature, ESR, Alpha1Antitrypsin values didn’t discriminate acute pancreatitis as mild or severe disease. Although those values found high in severe acute pancreatitis, mean 95% confidence limits of mild and severe attacks were overlapped throughout the study.

Of the inflammatory markers studied, CRP was able to differentiate acute pancreatitis into mild and severe forms with greatest precision.

PATIENT WITH ACUTE PANCREATITIS

PANCREATIC NECROSIS



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PATIENT CONSENT FORM

STUDY TITLE

STUDY ON C REACTIVE PROTEIN – AN AID FOR ASSESSING AND MONITORING THE SEVERITY OF ACUTE PANCREATITIS IN GMKMCH SALEM

Department of General surgery , GMKMCH SALEM

PARTICIPANT NAME : AGE : SEX:

I.P. NO :

I confirm that I have understood the purpose of investigation/surgical procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible out comes that may occur during and after providing nutrition. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study for various investigation /surgical procedures and their outcomes.

Date /

Time : Signature / Thumb Impression Of Patient

Place :

Patient name :

Signature of the investigator:

Name of the investigator :

PROFORMA

NAME:

ADDRESS:

AGE/SEX:

RELIGION:

O.PNo:

I.P No:

D.O.A:

TIME & DATE OF OPERATION:

D.O.D:

B. CHIEF COMPLAINTS:

C.**PAST HISTORY:**

1. DM : Yes/ No
2. TB : Yes/ No
3. CARDIAC DISEASE
4. BRONCHIAL ASTHMA
5. PREVIOUS SURGERY,PREVIOUS H/O ACUTE PANCREATITIS
6. JAUNDICE
7. CIRRHOSIS

D.**PERSONAL HISTORY:**

SMOKER

ALCOHOLIC

E.**INITIAL ASSESSMENT OF PATIENT**

1.VITALS:

PR :

BP :

RR :

Temperature :

2.GENERAL SIGNS:

Pallor

Tongue

Skin

Icterus

Cyanosis

Lymphadenopathy:

F. Assessment of abdomen

1)INSPECTION:

a) shape of the abdomen
b)skin
c)umbilicus
d)movement of the abdomen –respiratory ,peristaltic
e)swelling
f)Hernial orifices

2) PALPATION

a)Temperature
b)Tenderness
c)Distension
d)Palpation of mass
e)Palpation of abdominal organs
f)Muscle guard
g)Palpation of hernia sites

3)**PERCUSSION**

4)AUSCULTATION

Bowel sounds

G) Examination of left supraclavicular lympnodes

H) EXTERNAL GENITALIA

I) PER RECTAL EXAMINATION

J) SYSTEMIC EXAMINATION:

CVS

RS

CNS

MUSCULO SKELETAL SYSTEM

CLINICAL DIAGNOSIS

INVESTIGATIONS

A. COMPLETE BLOOD PICTURE – HB, PCV,TC,DC,ESR,RBC,PLATE LET

B. BLOOD GROUPING & TYPING

C. BT/CT

D. SERUM AMYLASE ,SERUM LIPASE, C-REACTIVE PROTEIN – TIME OF ADMISSION ,EVERY 48 HRS TILL THE LEVEL DECREASES

E. ANTIPROTEASE:ALPHA1 ANTITRYPSIN ALONG WITH CRP

F. HIV

G. ECG

H. URINE: Macro

Micro

Albumin

Sugar

I. BLOOD:

RBS

BLOOD UREA

SER.CREATININE

J. CHEST X RAY PA VIEW :
X –RAY ABDOMEN ERECT:

K. ABDOMEN & PELVIS USG
C.T ABDOMEN-PLAIN /CONTRAST

DIAGNOSIS:

OUTCOME OF THE TREATMENT:

1.IMPROVEMENT

2.WORSENING OF DISEASE

3.MULTIORGAN FAILURE

4.NEEDING SURGICAL INTERVENTION – PSEUDOCYST, NECROSECTOMY

5.CHRONIC PANCREATITIS

6.DEATH

MEDICAL / SURGERY:

S.NO	Name	AGE	IP No	MS	ESR VALUES (mm / Hour)					
					DAY-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
1	Perumal	35	47048	1	34	28	20	16	10	12
2	Vijay	35	51792	1	30	24	20	18	10	10
3	Murugaiya	55	54678	1	34	26	20	16	10	10
4	Karthi	40	56582	2	60	50	40	38	28	22
5	Saravanan	30	58444	1	30	28	22	16	10	14
6	Iyappan	28	60936	1	28	24	28	14	10	12
7	Peraman	43	19115	1	32	30	26	20	11	10
8	Govindasamy	40	63640	2	68	52	48	34	30	26
9	Madesh	35	79736	2	70	60	50	48	36	24
10	Selvamani	56	81720	2	82	64	48	36	28	16
11	Madesh	38	84358	1	30	28	20	18	14	10
12	Thangaraj	45	85698	2	56	48	50	40	32	28
13	Selvaraj	50	36118	1	30	24	20	18	16	10
14	Parameshwaran	35	86030	1	34	20	20	16	12	10
15	Jeevasubramanian	42	86370	1	36	26	26	18	14	12
16	Nagaraj	28	87968	1	40	32	28	16	10	8
17	Ilayaraja	36	97934	1	42	36	30	18	14	10
18	Chandrasekaran	40	89344	1	40	30	26	20	16	12
19	Jayaraman	35	88222	2	68	54	48	34	28	14
20	Om prakash	29	39010	1	34	24	30	16	14	12
21	Peter	45	91358	2	78	62	50	48	36	24
22	Raja	23	94090	1	30	24	20	18	16	10
23	Manivel	30	96052	2	60	58	60	52	48	32
24	Raju	45	96158	1	38	26	20	18	20	14
25	Loganathan	43	97715	2	78	60	58	42	30	18
26	David	25	25347	2	100	96				
27	Ramachandran	45	98320	1	30	26	22	18	14	10
28	Raja	45	99158	2	96	84	78	60	42	24
29	Arivalagan	44	99326	2	98	86	60	52	34	26
30	Sivasankar	19	100222	1	80	70	58	38	30	22
31	Ramesh	36	100308	2	74	70	68	48	34	20
32	Muthusamy	37	100684	2	60	58	48	40	32	24
33	Basha	35	101108	1	30	26	20	18	10	12
34	Muthukumar	14	100800	2	58	44	38	40	30	26
35	Kumar	42	102500	1	32	28	30	26	20	14
36	Velmurugan	25	103640	2	48	50	42	36	26	20
37	Murugesan	40	105182	1	41	30	24	20	18	16
38	Prakash	36	106336	1	34	28	24	20	18	16
39	Jayaprakash	35	110726	2	60	54	50	48	30	15
40	Sivasubramaniyan	32	110734	1	30	26	30	26	16	10

S.NO	Name	AGE	IP No	MS	ESR VALUES (mm / Hour)					
					DAY-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
41	Arumugam	65	113706	1	40	32	36	28	18	12
42	Anandan	27	115890	2	60	54	48	36	30	20
43	Govindan	50	118184	1	30	32	28	20	18	14
44	Eswaran	40	23	1	34	30	24	20	20	18
45	Abinesh	24	828	1	40	34	28	20	20	16
46	Raja	40	386	1	70	62	50	48	40	31
47	Raju	40	40	2	60	62	54	44	36	24
48	Sudhakaran	50	4081	1	40	36	26	22	20	18
49	Madesh	35	6502	1	40	36	34	28	16	14
50	Selvakumar	40	7760	2	68	54	56	44	30	26
51	Yesudass	45	8642	2	72	68	64	56	40	20
52	Arjunan	27	1030	2	74	68	70	52	38	26
53	Thamaraikannan	27	9421	2	80	70	62	48	36	24
54	Shanmugam	43	11000	1	34	30	28	24	20	18
55	Rajivgandhi	32	12345	2	70	72	62	54	38	24
56	Murugan	50	14869	1	32	30	28	24	20	18
57	Murugesan	40	18034	1	40	30	28	20	18	14
58	Thangavel	60	22264	1	40	32	28	20	16	10
59	Raman	37	22300	2	68	70	64	56	48	38
60	Venkatesh	26	22579	1	38	34	30	28	18	16
61	Murugan	40	26890	2	78	74	68	50	46	30
62	Arumugam	36	26964	1	36	30	34	24	20	18
63	Vadivel	37	27048	1	34	38	32	36	24	20
64	Govindan	65	29541	2	70	68	58	42	38	26
65	Siva	30	29565	2	68	60	52	44	32	24
66	Kannan	31	30817	1	40	36	32	30	24	14
67	Arumugam	36	30990	2	70	60	52	43	34	26
68	Karupannan	65	34761	2	78	62	54	40	30	20
69	Tamilvannan	42	33581	2	70	62	50	40	30	20
70	Kuppusamy	45	34764	2	80	64	50	42	34	24
71	Dhamodharan	25	38129	2	72	64	52	42	34	24
72	Muthukaruppan	36	42136	2	88	68	54	44	36	24
73	Mani	40	32200	2	70	62	50	38	28	24
74	Vasanthakumar	45	34155	2	80	74	64	58	42	34
75	Senthil	32	391042	1	38	28	30	24	20	16

S.NO	Name	AGE	IP No	Temperature (Farenheit)					
				DAY-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
1	Perumal	35	47048	98	98	98	98	98	98
2	Vijay	35	51792	98	98	98	98	98	98
3	Murugaiya	55	54678	98	98	98	98	98	98
4	Karthi	40	56582	100	101	100	98	98	98
5	Saravanan	30	58444	98	98	98	98	98	98
6	Iyappan	28	60936	99	99	99	98	98	98
7	Peraman	43	19115	99	99	99	98	98	98
8	Govindasamy	40	63640	98	98	98	98	98	98
9	Madesh	35	79736	98	98	98	98	98	98
10	Selvamani	56	81720	100	102	101	99	98	98
11	Madesh	38	84358	98	98	98	98	98	98
12	Thangaraj	45	85698	100	101	99	98	98	98
13	Selvaraj	50	36118	98	98	98	98	98	98
14	Parameshwaran	35	86030	98	98	98	98	98	98
15	Jeevasubramanian	42	86370	98	98	98	98	98	98
16	Nagaraj	28	87968	99	100	99	98	98	98
17	Ilayaraja	36	97934	98	98	98	98	98	98
18	Chandrasekaran	40	89344	98	98	98	98	98	98
19	Jayaraman	35	88222	101	100	99	98	98	98
20	Om prakash	29	39010	98	98	98	98	98	98
21	Peter	45	91358	101	100	99	98	98	98
22	Raja	23	94090	98	98	98	98	98	98
23	Manivel	30	96052	100	100	99	98	98	98
24	Raju	45	96158	98	100	99	98	98	98
25	Loganathan	43	97715	98	98	98	98	98	98
26	David	25	25347	102	101				
27	Ramachandran	45	98320	98	98	98	98	98	98
28	Raja	45	99158	99	100	99	98	98	98
29	Arivalagan	44	99326	98	98	98	98	98	98
30	Sivasankar	19	100222	98	98	98	98	98	98
31	Ramesh	36	100308	98	98	98	98	98	98
32	Muthusamy	37	100684	100	99	99	98	98	98
33	Basha	35	101108	98	98	98	98	98	98
34	Muthukumar	14	100800	100	101	100	99	98	98
35	Kumar	42	102500	98	98	98	98	98	98
36	Velmurugan	25	103640	98	98	98	98	98	98
37	Murugesan	40	105182	100	100	99	98	98	98
38	Prakash	36	106336	100	99	99	98	98	98
39	Jayaprakash	35	110726	99	99	98	98	98	98
40	Sivasubramaniyan	32	110734	98	98	98	98	98	98

S.NO	Name	AGE	IP No	Temperature (Farenheit)					
				DAY-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
41	Arumugam	65	113706	98	98	98	98	98	98
42	Anandan	27	115890	98	98	98	98	98	98
43	Govindan	50	118184	98	98	98	98	98	98
44	Eswaran	40	23	98	98	98	98	98	98
45	Abinesh	24	828	98	98	98	98	98	98
46	Raja	40	386	100	100	99	98	98	98
47	Raju	40	40	100	99	99	98	98	98
48	Sudhakaran	50	4081	98	98	98	98	98	98
49	Madesh	35	6502	98	98	98	98	98	98
50	Selvakumar	40	7760	101	100	99	99	98	98
51	Yesudass	45	8642	101	100	99	98	98	98
52	Arjunan	27	1030	98	98	98	98	98	98
53	Thamaraikannan	27	9421	100	100	99	98	98	98
54	Shanmugam	43	11000	98	98	98	98	98	98
55	Rajivgandhi	32	12345	100	101	100	98	98	98
56	Murugan	50	14869	98	98	98	98	98	98
57	Murugesan	40	18034	98	98	98	98	98	98
58	Thangavel	60	22264	99	99	99	98	98	98
59	Raman	37	22300	100	101	99	98	98	98
60	Venkatesh	26	22579	98	98	98	98	98	98
61	Murugan	40	26890	98	98	98	98	98	98
62	Arumugam	36	26964	98	98	98	98	98	98
63	Vadivel	37	27048	98	98	98	98	98	98
64	Govindan	65	29541	98	98	98	98	98	98
65	Siva	30	29565	100	100	98	98	98	98
66	Kannan	31	30817	98	98	98	98	98	98
67	Arumugam	36	30990	98	98	98	98	98	98
68	Karupannan	65	34761	100	100	98	98	98	98
69	Tamilvannan	42	33581	100	100	98	98	98	98
70	Kuppusamy	45	34764	98	98	98	98	98	98
71	Dhamodharan	25	38129	100	99	98	98	98	98
72	Muthukaruppan	36	42136	101	100	99	98	98	98
73	Mani	40	32200	98	98	98	98	98	98
74	Vasanthakumar	45	34155	98	98	98	98	98	98
75	Senthil	32	391042	98	98	98	98	98	98

S.NO	Name	AGE	IP No	Alpha 1 Anti Trypsin (gm/Ltr)					
				Day-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
1	Perumal	35	47048	3	3	3.2	3.2	2	2
2	Vijay	35	51792	3	3.2	3.2	3.2	2.2	2
3	Murugaiya	55	54678	3	3	3.2	3.2	2	2
4	Karthi	40	56582	4	4	3.6	3.6	3	3
5	Saravanan	30	58444	3	3.2	3.2	3.2	2	2
6	Iyappan	28	60936	3.2	3.2	3.2	3	2.2	2.2
7	Peraman	43	19115	3	3	3	2.4	2.4	2
8	Govindasamy	40	63640	4.2	4.2	4	3.2	3.2	2.4
9	Madesh	35	79736	4.4	4	4	4	3.2	3
10	Selvamani	56	81720	4.8	4.2	4	3.2	3	3
11	Madesh	38	84358	3	3.2	3.2	3.2	2	2
12	Thangaraj	45	85698	3.8	4	4	3.6	3	3
13	Selvaraj	50	36118	3	3	2.4	2.2	2.2	2
14	Parameshwaran	35	86030	3	3	2.4	2	2	2
15	Jeevasubramanian	42	86370	3.2	3.2	3	2.4	2.4	2
16	Nagaraj	28	87968	3.6	3.4	3.4	2	2	1.8
17	Ilayaraja	36	97934	3.6	3.4	3.4	2	2	2
18	Chandrasekaran	40	89344	3.6	3	3	2.4	2.4	2
19	Jayaraman	35	88222	4.2	4	4	3.4	3	3
20	Om prakash	29	39010	3	3	3	2	2	1.4
21	Peter	45	91358	4.6	4	4	3	2.6	2
22	Raja	23	94090	3	2.8	2.8	2	2	2
23	Manivel	30	96052	4	4	4	3	2.4	2
24	Raju	45	96158	3.2	3	3	2.2	2.2	2
25	Loganathan	43	97715	4.6	4	4.4	3.4	3	3
26	David	25	25347	5.6	5.4				
27	Ramachandran	45	98320	3	3	2.4	2	2	2
28	Raja	45	99158	4.8	4.6	4.6	4	4	3.2
29	Arivalagan	44	99326	4.8	4.6	4.6	2	4	3.2
30	Sivasankar	19	100222	4.6	4.4	4.2	3.6	3.6	3
31	Ramesh	36	100308	4.2	4	4	3.6	3	3
32	Muthusamy	37	100684	4	4	4	3.2	3	3
33	Basha	35	101108	3	3	2.4	2	2	2
34	Muthukumar	14	100800	4	3.4	3.2	2.6	2	2
35	Kumar	42	102500	3	3	2.4	2	2	2
36	Velmurugan	25	103640	4	4	4	3.2	3	3
37	Murugesan	40	105182	3.2	3	2.6	2.6	2	2
38	Prakash	36	106336	3	3	2.4	2.4	2	2
39	Jayaprakash	35	110726	4.4	4.4	4	4	3.2	3
40	Sivasubramaniyan	32	110734	3	3	3	3	2	2

S.NO	Name	AGE	IP No	Alpha 1 Anti Trypsin (gm/Ltr)					
				Day-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
41	Arumugam	65	113706	3.6	3.6	3	3	2.3	2.2
42	Anandan	27	115890	4.4	4.4	4	3	3	2.4
43	Govindan	50	118184	3	3	3	2.4	2	2
44	Eswaran	40	23	3	3	2.6	2	2	2
45	Abinesh	24	828	3.4	3.3	3	2.2	2	2
46	Raja	40	386	4.6	4	4	4	3.4	3
47	Raju	40	40	4	4	4	3.6	3	2.6
48	Sudhakaran	50	4081	3.6	3.4	3	3	2	2
49	Madesh	35	6502	3.6	3.4	3	3	2.2	2
50	Selvakumar	40	7760		3.2	2.8	2.2	1	1
51	Yesudass	45	8642	4.6	4.2	4	4	3.6	3
52	Arjunan	27	1030	4.6	4.4	4.4	4	3.2	3
53	Thamaraikannan	27	9421	4.8	4.6	4.6	4	3	3
54	Shanmugam	43	11000	3	3	2.6	2.6	2	2
55	Rajivgandhi	32	12345	4.6	4.6	4	4	3.6	3.5
56	Murugan	50	14869	3	3	3	2.6	2.2	2
57	Murugesan	40	18034	3	3	3	2.4	2	2
58	Thangavel	60	22264	3	3	3	2.4	2	2
59	Raman	37	22300	4.2	4.2	4	4	3	3
60	Venkatesh	26	22579	3.2	3	3	2.6	2	2
61	Murugan	40	26890	4.8	4.6	4.6	4	3.5	3
62	Arumugam	36	26964	3	3	3	2.4	2	2
63	Vadivel	37	27048	3	3	3	3	2.5	2.5
64	Govindan	65	29541	4.4	4.4	4.2	3.6	1.6	1.2
65	Siva	30	29565	4.1	4	4	3.5	3	3
66	Kannan	31	30817	3	3	2.7	2	2	1.2
67	Arumugam	36	30990	4.4	4.2	4.2	4	3	3
68	Karupannan	65	34761	4.6	4.4	4.4	3.5	3	3
69	Tamilvannan	42	33581	4.4	4.2	4	3.2	3	2
70	Kuppusamy	45	34764	4.8	4.4	4	3.8	3.2	2.5
71	Dhamodharan	25	38129	4.4	4	4	3.6	3.2	3
72	Muthukaruppan	36	42136	4.8	4.6	4	4	3.5	3
73	Mani	40	32200	4.4	4	4	3	2.8	2
74	Vasanthakumar	45	34155	4.8	4.8	4	4	3.2	3
75	Senthil	32	391042	3	3	2.5	2.4	2	1.6

S.NO	Name	AGE	IP No	C -Reactive Protein (mg/Ltr)					
				Day-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
1	Perumal	35	47048	58	60	54	40	24	10
2	Vijay	35	51792	55	65	58	32	12	8
3	Murugaiya	55	54678	42	40	44	30	10	6
4	Karthi	40	56582	98	190	172	140	100	60
5	Saravanan	30	58444	40	68	52	33	18	10
6	Iyappan	28	60936	52	76	68	38	18	8
7	Peraman	43	19115	54	96	100	84	42	12
8	Govindasamy	40	63640	102	132	121	100	92	68
9	Madesh	35	79736	99	142	104	98	83	75
10	Selvamani	56	81720	158	170	132	84	86	58
11	Madesh	38	84358	52	48	32	20	14	8
12	Thangaraj	45	85698	106	174	153	92	69	40
13	Selvaraj	50	36118	38	40	34	22	16	8
14	Parameshwaran	35	86030	44	63	52	40	22	6
15	Jeevasubramanian	42	86370	50	42	32	26	14	6
16	Nagaraj	28	87968	42	68	50	34	18	8
17	Ilayaraja	36	97934	40	52	38	23	18	10
18	Chandrasekaran	40	89344	38	46	30	25	16	8
19	Jayaraman	35	88222	120	152	138	82	60	52
20	Om prakash	29	39010	46	64	50	34	20	6
21	Peter	45	91358	110	176	142	98	64	38
22	Raja	23	94090	50	64	44	30	22	10
23	Manivel	30	96052	118	142	102	94	78	60
24	Raju	45	96158	63	74	48	23	11	6
25	Loganathan	43	97715	84	113	90	63	40	22
26	David	25	25347	164	192				
27	Ramachandran	45	98320	38	58	40	36	24	8
28	Raja	45	99158	114	134	120	94	78	60
29	Arivalagan	44	99326	126	132	102	90	71	62
30	Sivasankar	19	100222	78	82	64	48	30	21
31	Ramesh	36	100308	138	150	120	89	62	48
32	Muthusamy	37	100684	144	164	140	109	84	30
33	Basha	35	101108	44	30	24	14	10	6
34	Muthukumar	14	100800	122	144	120	99	77	48
35	Kumar	42	102500	50	56	42	28	16	8
36	Velmurugan	25	103640	100	142	138	109	74	42
37	Murugesan	40	105182	62	70	44	28	18	10
38	Prakash	36	106336	60	68	42	30	18	6
39	Jayaprakash	35	110726	78	94	84	69	55	30
40	Sivasubramaniyan	32	110734	38	40	30	18	10	6

S.NO	Name	AGE	IP No	C -Reactive Protein (mg/Ltr)					
				Day-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
41	Arumugam	65	113706	42	50	40	28	16	8
42	Anandan	27	115890	124	133	118	89	70	60
43	Govindan	50	118184	48	38	40	28	12	6
44	Eswaran	40	23	36	28	30	12	10	4
45	Abinesh	24	828	44	38	40	30	22	10
46	Raja	40	386	62	78	60	44	30	24
47	Raju	40	40	122	140	123	109	62	48
48	Sudhakaran	50	4081	48	60	40	24	10	6
49	Madesh	35	6502	42	50	40	32	18	6
50	Selvakumar	40	7760	120	140	110	99	80	60
51	Yesudass	45	8642	120	142	124	99	66	58
52	Arjunan	27	1030	114	120	110	99	88	62
53	Thamaraikannan	27	9421	106	130	100	89	82	50
54	Shanmugam	43	11000	46	50	40	28	18	6
55	Rajivgandhi	32	12345	118	132	110	89	70	40
56	Murugan	50	14869	46	40	32	24	18	6
57	Murugesan	40	18034	50	58	48	24	16	10
58	Thangavel	60	22264	48	60	40	28	18	8
59	Raman	37	22300	110	132	100	79	64	40
60	Venkatesh	26	22579	62	60	58	40	20	6
61	Murugan	40	26890	138	142	130	109	70	40
62	Arumugam	36	26964	50	30	42	30	21	10
63	Vadivel	37	27048	46	50	30	28	14	6
64	Govindan	65	29541	98	110	120	79	80	62
65	Siva	30	29565	136	140	110	89	68	48
66	Kannan	31	30817	46	54	40	30	18	10
67	Arumugam	36	30990	84	98	100	79	64	26
68	Karupannan	65	34761	120	140	118	99	62	56
69	Tamilvannan	42	33581	80	100	84	79	47	36
70	Kuppusamy	45	34764	94	110	98	79	52	48
71	Dhamodharan	25	38129	136	140	126	99	62	52
72	Muthukaruppan	36	42136	130	148	100	89	66	50
73	Mani	40	32200	94	100	96	79	60	42
74	Vasanthakumar	45	34155	96	98	100	79	42	30
75	Senthil	32	391042	60	40	38	28	16	9

S.NO	Name	AGE	IP No	White cell count (Cells / Cu.mm)					
				Day-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
1	Perumal	35	47048	12000	12200	12200	11600	11400	11400
2	Vijay	35	51792	11000	11200	11200	10600	10400	10400
3	Murugaiya	55	54678	10800	10600	10600	9800	9600	9600
4	Karthi	40	56582	14200	14000	14000	13600	13400	13400
5	Saravanan	30	58444	11000	11200	11200	10600	10200	10200
6	Iyappan	28	60936	10800	10600	10600	10000	10200	10200
7	Peraman	43	19115	11000	11200	11200	10600	10400	10400
8	Govindasamy	40	63640	14600	14400	14400	14000	14000	13800
9	Madesh	35	79736	13600	13400	13400	12100	12000	12000
10	Selvamani	56	81720	14300	14200	14200	13200	13000	13000
11	Madesh	38	84358	10000	10200	10200	9800	9600	9600
12	Thangaraj	45	85698	13000	12800	12800	12000	12000	12000
13	Selvaraj	50	36118	7800	8000	8000	7400	7500	7500
14	Parameshwaran	35	86030	9600	9700	9700	10000	10200	10200
15	Jeevasubramanian	42	86370	11000	11200	11200	12000	12100	12000
16	Nagaraj	28	87968	9800	9800	10000	11100	11200	11200
17	Ilayaraja	36	97934	11000	11200	11200	12000	12200	12200
18	Chandrasekaran	40	89344	11000	11100	11100	12800	12800	12600
19	Jayaraman	35	88222	14400	14600	14600	12000	12000	12000
20	Om prakash	29	39010	12000	12200	12200	11800	11600	11600
21	Peter	45	91358	13600	13800	12600	12400	12400	12400
22	Raja	23	94090	11000	11200	11200	10600	10400	10400
23	Manivel	30	96052	14600	14800	14800	13200	13000	13000
24	Raju	45	96158	11000	11200	11200	10800	10600	10600
25	Loganathan	43	97715	13800	14000	14000	13000	13000	13000
26	David	25	25347	15400	15200				
27	Ramachandran	45	98320	10200	10400	10400	9800	9600	9600
28	Raja	45	99158	14800	14600	14600	12800	13000	13000
29	Arivalagan	44	99326	14600	14400	14400	13800	13600	13600
30	Sivasankar	19	100222	13800	13400	13600	12800	12600	12500
31	Ramesh	36	100308	14000	14200	14200	13800	13600	13600
32	Muthusamy	37	100684	13600	13800	13800	12600	12400	12400
33	Basha	35	101108	11000	10600	10600	9400	9600	9600
34	Muthukumar	14	100800	12800	12600	12600	11800	11600	11600
35	Kumar	42	102500	11400	11200	11200	10800	10800	10800
36	Velmurugan	25	103640	13200	13400	13400	12800	12600	12600
37	Murugesan	40	105182	10400	10200	10200	9600	9800	9800
38	Prakash	36	106336	11200	11400	11400	10800	10600	10600
39	Jayaprakash	35	110726	13200	13400	13400	12800	12400	12400
40	Sivasubramaniyan	32	110734	9800	9600	9600	8600	8400	8400

S.NO	Name	AGE	IP No	White cell count (Cells / Cu.mm)					
				Day-1	DAY-3	DAY-5	DAY-7	DAY-9	DAY-11
41	Arumugam	65	113706	10600	10400	10400	9800	9600	9600
42	Anandan	27	115890	11400	11600	11600	10800	10600	10600
43	Govindan	50	118184	10400	10200	10200	9800	9600	9600
44	Eswaran	40	23	9800	9600	9600	8800	8600	8600
45	Abinesh	24	828	10200	10000	10000	9800	9600	9600
46	Raja	40	386	11200	11400	11400	10800	10600	10600
47	Raju	40	40	13200	13400	13400	13000	12800	12800
48	Sudhakaran	50	4081	9600	9800	9600	9000	8800	8600
49	Madesh	35	6502	11000	11200	11200	10800	10800	10800
50	Selvakumar	40	7760	14900	14800	14800	13000	13200	13200
51	Yesudass	45	8642	14600	14400	14400	13600	13000	13000
52	Arjunan	27	1030	13600	13400	13400	12600	12800	12800
53	Thamaraikannan	27	9421	14200	14400	14400	13800	13600	13600
54	Shanmugam	43	11000	11000	11200	11200	11000	10800	10800
55	Rajivgandhi	32	12345	13600	13800	13800	12800	12600	12600
56	Murugan	50	14869	11000	11200	11200	10800	10600	10600
57	Murugesan	40	18034	10800	10600	10600	10000	10200	10200
58	Thangavel	60	22264	9800	9600	9600	9000	9200	9200
59	Raman	37	22300	14200	14400	14400	13800	13600	13600
60	Venkatesh	26	22579	11800	11600	11600	10800	10600	10600
61	Murugan	40	26890	14200	14000	14000	13800	13600	13600
62	Arumugam	36	26964	9800	9600	9600	8800	8600	8600
63	Vadivel	37	27048	9600	9800	9800	8800	8600	8600
64	Govindan	65	29541	14600	14800	14800	13800	13600	13600
65	Siva	30	29565	13800	13600	13600	13000	12800	12800
66	Kannan	31	30817	12000	12800	12800	11000	11200	11200
67	Arumugam	36	30990	13800	13600	13600	13100	12900	12900
68	Karupannan	65	34761	14200	14000	14000	13600	13800	13600
69	Tamilvannan	42	33581	13800	13600	13600	12800	12600	12600
70	Kuppusamy	45	34764	14400	14200	14300	13600	13400	13400
71	Dhamodharan	25	38129	13600	13400	13400	12800	12600	12400
72	Muthukaruppan	36	42136	14000	14200	14200	13600	13400	13400
73	Mani	40	32200	14300	14200	14200	13800	13600	13700
74	Vasanthakumar	45	34155	13600	13400	13300	13000	12600	12600
75	Senthil	32	391042	10000	10200	10200	9800	9600	9600